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MANUAL OF
DISEASES OF WOMEN

HENRY SCHMITZ, A.M., M.D., F.A.C.S.

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MANUAL

OF

DISEASES OF WOMEN

BY

HENRY SCHMITZ, A.M., M.D., F.A.C.S.

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Repetitio est mater studiorum.

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RESPECTFULLY DEDICATED TO

DR. ALBERT J. OCHSNER

AS AN APPRECIATION OF HIS UNSELFISH AND INSPIRING PERSONAL FRIENDSHIP.

PREFACE

This volume was written on the numerous requests of students attending the author's lectures and clinics at the Chicago College of Medicine and Surgery.

The arrangement of the subjects and the description of the various gynecologic diseases are based on the author's translation of A. Martin's *"Pathology and Treatment of Diseases of Women"* published in 1912.

Illustrations have not been inserted in the text. The student is referred to other books on gynecology for the study of pathological and clinical illustrations, especially the following: Martin-Schmitz, *Pathology and Treatment of Diseases of Women;* Kelly, *Medical and Operative Gynecology;* Menge-Opitz, *Handbuch der Frauenheilkunde;* Ashton, *Gynecology;* Crossen, *Diseases of Women and Operative Gynecology;* Döderlein-Krönig, *Operative Gynäkologie;* Ochsner and Percy, *New Manual of Surgery.* Students intending to do more advanced work are requested to read Veit, *Handbuch der Gynäkologie* and Liepman, *Handbuch der Frauenheilkunde.*

Operative procedures also have not been discussed or described except those that are not usually demonstrated in a gynecological clinic on account of the rarity of their necessity. Special stress has been laid on the pathology, the diagnosis and the indications for treatment.

The author sincerely hopes that the book will prove of value to the student, be an aid to the teacher and a welcome adviser to the general practitioner.

In conclusion thanks are expressed to my associates in the college gynecological department, and particularly Dr. L. C. Borland, for valuable advice.

HENRY SCHMITZ.

Chicago, January, 1916.

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MANUAL OF
DISEASES OF WOMEN

CHAPTER I.

GYNECOLOGICAL EXAMINATION.

A gynecological examination comprises the taking of an exact history and a careful physical exploration. The latter should include a general physical examination of the entire body and a special physical examination of the abdominal wall, the abdominal and pelvic organs, the bladder, ureters, rectum, and the pelvic outlet.

To be able to render an exact diagnosis it is imperative to be systematic in the procedure of an examination. The following outline will facilitate the taking of a comprehensive history and give the steps of the physical examination.

Case number, date of entrance, service of Dr. .....
..... Referred by Dr. .....

Name of patient, residence, telephone number, age, nationality, civil state, name of husband, father, nearest relative or friend of patient, business or profession of same, name of person responsible for the fee.

Present illness: Date of onset and duration of the presenting symptoms and an exact statement from the patient.

Previous condition and illnesses, including diseases of childhood or any other abnormal condition.

Social condition and habits, including personal habits, social conditions and surroundings.

Family history: (Details).

Menstrual history: The beginning and termination of

the menstrual life. Periodicity, duration, character and quantity of flow. Menstrual pain; time of its occurrence, duration, location and character. Constitutional symptoms, as headache, backache, lassitude, weakness and so forth.

Intermenstrual discharges: Character, quantity, time of their occurrence and duration.

Intermenstrual pain: Character, location, time of occurrence and duration.

Maternal history: Number and dates of pregnancies.

Abortions: Number of same, causes, dates of occurrence and period of gestation when they took place.

Character of pregnancies, labors, puerperii and lactation periods.

Urinary system: Painful and frequent urinations, night urinations, amount, occurrence of pain, whether before, during or after micturition, incontinence of urine and so forth.

Alimentary system: Functions of stomach and bowels. Character and regularity of stools.

Respiratory and circulatory system: (Details).

Nervous system: (Details).

The general physical examination should consider the general appearance of the patient, including height, weight, condition of the skin, and examination of the head, neck, chest, abdomen and extremities.

The special physical examination should include: 1. Inspection, palpation, percussion, auscultation and mensuration of the abdomen.

2. Direct and indirect inspection of the external genitalia, vagina and cervix.

3. Bimanual palpation noting (a) position, (b) mobility, (c) size and form, (d) consistency and (e) sensitiveness of the uterus, tubes, ovaries, perimetrium and parametrium.

4. Examination of the bladder and rectum by inspec-

tion with the aid of the cystoscope and proctoscope, and by palpation.

5. Urinanalysis.

6. Bloodanalysis.

7. Microscopical and bacteriological examinations of tissues and discharges.

A diagnosis, even a tentative one, and an outline of the treatment should be recorded at the first examination. One is thus enabled to recognize mistakes made, to investigate the reasons for the same and also to observe the results of the treatment instituted.

The special examination. Preliminary considerations: The normal position of the uterus in a woman standing erect, with bladder and rectum empty, is almost horizontal. The fundus points to the upper border of the symphysis pubis. The posterior surface of the corpus (body) is directed upward, while the axis of the body points backward to the sacral promontory. The cervix is directed backward and downward, crossing the axis of the vagina nearly perpendicularly. The vagina is directed from below upwards and backwards.

This normal position is subject to variations within normal physiological limits resulting from (a) changes in the position of the woman, (b) distension of the bladder with urine and (c) accumulation of feces in the rectum.

1. In the recumbent position the vagina is directed downwards and backwards, the cervix pointing upwards and forwards and the corpus directly forward towards the anterior abdominal wall.

2. A distended bladder lifts the fundus of the uterus upwards and pushes the entire organ backwards, causing a retro-position and an elevation of the uterus.

3. A distended rectum pushes the uterus forwards and upwards, causing an ante-position and an elevation of the organ.

If the bladder or rectum be emptied the uterus reassumes its normal position. These deviations from the typical position of the uterus may be regarded as physiological. *It is evident that the bladder and rectum must be thoroughly evacuated before a gynecological examination is attempted.*

The special pelvic examination comprises: 1. Inspection, palpation, percussion, auscultation, and at times mensuration of the anterior abdominal wall and abdominal cavity.

2. Inspection and palpation of the vulva, noting (a) the clitoris, (b) the meatus urinarius, (c) the hymen, (d) Bartholin's gland, (e) anterior vaginal wall, (f) posterior vaginal wall and perineum, (g) anus and (h) the condition of the muscles of the pelvic outlet.

3. A combined bimanual examination of the pelvic organs.

4. An inspection of the vaginal walls and cervix by the use of a vaginal speculum.

5. Sounding of the uterine cavity.

6. Dilation of the cervical canal and palpation of the uterine cavity.

7. Curettage of the uterus and excision of pieces from the cervix for microscopical diagnosis.

8. Bacteriological examination.

9. Cystoscopical examination.

10. Proctoscopical examination.

Let us discuss in detail examinations mentioned under 3, 4, 5, 6 and 7.

3. A *combined bimanual examination* means the palpation of the internal organs by one or more fingers introduced into the vagina, with simultaneous counterpressure on the organs through the relaxed and invaginated anterior abdominal wall by the other hand. In this way we determine, 1. position, 2. movability, 3. size

and form, 4. consistency and 5. sensitiveness of the organs.

Technic. Always place the patient in the same position, which is usually the recumbent. Other postures which may be necessary at times are the left lateral, or Sims', the standing and knee-chest position. In the recumbent position the patient is put flatly on her back. The lower extremities are bent at the knee and the thighs flexed on the abdomen and secured in stirrups. The knees must be widely separated to insure relaxation of the anterior abdominal wall. Waist-bands and corsets must be removed. The patient should be draped. The examining physician should stand directly in front of the patient. He, thereby, prevents the patient from bringing the knees in apposition. The fingers to be inserted in the vagina should be those of the left hand, and they should be protected by sterile rubber gloves. The physician should place his left foot on a foot-stool and rest his left elbow on his left thigh. This arrangement allows the greatest possible freedom of the left hand and forearm.

Rules for combined palpation. (a) Locate the cervix and determine its position, movability, size, form, consistency, and sensitiveness. (b) Locate the uterus by bimanual efforts and use it as a land-mark for further examination and then determine the position, movability, size, form, consistency and sensitiveness of the organ. (c) Next locate the adnexa (appendages) which are situated laterally and posteriorly to the uterus, and determine the position, movability, size, form, consistency and sensitiveness of same. *Rule: The lighter the touch and the less force used in advancing fingers in the vaginal vault, and in invaginating the abdominal wall, the sooner the purpose of the examination will be attained and the more distinctly can the parts be felt.* (d) The results of the vagino-abdominal examination should in-

variably be verified by a combined rectal examination. The gloved index finger of the left hand is inserted into the rectum. The perineum is pressed upward as far as necessary. Counter-pressure on the organs is made through the pelvic inlet as in vagino-abdominal palpation. (e) A recto-abdominal examination alone should only be insisted upon in virgins and women having inaccessible or impermeable vaginas. (f) A general anesthetic should be used in patients who cannot be persuaded to relax, if for only a moment, their board-like, tense abdominal muscles. Such occasions, however, arise very rarely.

4. The bimanual examination should be followed by an *inspection of the cervix and vagina,* which are exposed to view by means of (a) bivalve, self-retaining speculum, (b) tubular speculum or (c) vaginal retractors.

5. *Sounding.* The information gained by sounding the uterine cavity is: (a) the length of the cervix and cavity of the uterus; (b) the condition of the internal os; (c) the width of the uterine cavity; (d) the position of the uterus and (e) the existence of irregularities, small tumors and so forth of the endometrium.

Contra-indications to sounding of the uterine cavity are: (a) the slightest suspicion of pregnancy or a history of amenorrhea, (b) acute or subacute peri- or parametritis, (c) acute inflammation of the uterus and menstruating or bleeding uteri, (d) infection of the vulva and vagina.

The rules of the *technic* to be followed are: (a) Sounds must be employed only under the strictest aseptic precautions. (b) Force must never be used in inserting a sound. (c) If pain is caused by the procedure desist at once from further instrumentation.

The *danger* of the operation consists in a perforation of the uterus. Sounding is a procedure which should

only be employed by a skillful surgeon, and the more skillful he is the less will be the necessity of its use. Considering the dangers anent its use, sounding should be omitted from ordinary gynecological examinations.

6. *Dilation of the cervical canal and palpation of the uterine cavity. Indications:* (a) To determine diseased conditions of the endometrium, as cancer, mucous polypi. (b) To remove remnants of abortion or labor. (c) To facilitate the diagnosis of some uterine tumors. The *contra-indications* are the same as for sounding.

Technic. Anesthesia should be used if necessary. Strict aseptic precautions must be insisted upon. The cervix should be exposed through a speculum, fixed by a tenaculum forceps and dilated with Hegar's sounds until the desired dilatation is reached.

The *dangers* are: (a) Lacerations of the cervix and (b) perforation of the uterus. They are minimized by the use of Hegar's sounds, while the use of Goodell's dilator is directly dangerous, leading always to a loss of continuity of surface epithelium and often to extensive laceration.

7. *Curettage of the uterus.* The *indications* are: 1. Therapeutic: (a) To arrest hemorrhage, (b) to remove the endometrium, (c) to dislodge retained tissues. 2. Diagnostic: To exclude or confirm malignancy.

The *contra-indications* are the same as for sounding. The steps of the *technic* are: (a) general anesthesia if necessary, (b) strict aseptic precautions, (c) dilation of the cervical canal, (d) the curetting, (e) microscopical examination of the removed tissues, using a freezing microtome. If malignancy is present other surgical procedures must be immediately instituted. (f) Cauterization of the endometrium.

Remarks: Force must never be used in inserting the curette. Curettage should not be followed by intrauterine irrigation, but dry gauze should be inserted, with

a cork-screw-like motion, into the uterine cavity to remove tissue débris.

Cauterization is necessary if it is advisable to destroy any remaining portions of the endometrium. In this event the uterine cavity is packed with a strip of gauze saturated in the cauterizing fluid, such as Monsell's solution, tincture of iodine, 50 per cent. aqueous solution of zinc chloride, to be followed by powdered sodium bicarbonate to neutralize the action of the zinc chloride, 95 per cent. phenol, neutralized by 95 per cent. alcohol, 4 to 40 per cent. solution of formalin. Following this it is well to pack the uterine cavity and vaginal canal with yellow gauze saturated with Beck's paste. The tampon should be removed on the third day following the operation.

Regeneration of the endometrium after curettage ensues from the crypts of tubular glands and becomes completed within three weeks. Menstruation generally reappears within six weeks. The *after treatment* consists of rest in bed for three or four days. Liquid diet is ordered as soon as the nausea following the anesthesia subsides.

Perforation of the uterus is due to soft uterine walls or injudicious force in inserting an instrument. The *treatment* of perforation is as follows: Remove the instrument and suspend all further manipulations. Do not pack the uterine cavity with a tampon. Put the patient to bed in the Fowler position. Apply an ice coil to the lower abdomen. If alarming symptoms do not occur within forty-eight hours, the patient is out of danger.

Diagnostic excision of tissues. At times a piece of tissue must be excised from the uterus and subjected to microscopical examination for purposes of diagnosis. To avoid hemorrhage after such an excision the vagina should be packed with iodoform gauze.

CHAPTER II.

GENERAL OUTLINE FOR MEMORIZING THE DIFFERENT DISEASES.

The application of the following outline will facilitate the study of diseases, medical or surgical. It will lead to a systematic preparation of quizzes and examinations. The student should memorize this outline by always applying it to his studies.

1. Synonyms.
2. Definition.
   a. Acute or chronic.
   b. Cause (if known).
   c. Characteristics.
      1. Anatomical.
      2. Clinical.
3. Etiology.
   a. Predisposing.
      1. Individual.
         a. Race.
         b. Sex.
         c. Age.
         d. Heredity.
         e. Previous diseases.
         f. Occupation.
      2. Surroundings.
         a. Climate.
         b. Season.
         c. Social conditions.

3. Etiology—continued.
    b. Exciting.
        1. Mechanical.
        2. Thermical.
        3. Parasitic.
        4. Pathological.
        5. Congenital.
        6. Circulatory.
        7. Inflammatory.
        8. Degenerative.
        9. New growth.
        10. Nervous.
        11. Bacterial.
            a. Name and synonym.
            b. Morphology.
                1. size.
                2. shape.
                3. grouping.
                4. capsule.
                5. spores.
                6. motility.
            c. Stains.
                1. ordinary.
                2. Gram.
                3. special.
            d. Culture.
                1. Physical condition.
                    a. air.
                    b. temperature.
                    c. reproduction.
                2. Media.
                    a. bouillon.
                    b. gelatin.
                        1. stab.
                        2. plate.

3. Etiology—continued.
    2. Media—continued.
        c. agar.
        d. potato.
        e. milk.
        f. blood serum.
        g. special media.
    e. Resistance.
        1. Hot and cold.
        2. Injury.
        3. Drugs.
    f. Distribution.
        1. Within the body.
        2. Without the body.
    g. Special reaction (Widal, Wasserman, compliment fixation, Abderhalden).
    h. Pathogenicity.
4. Morbid Anatomy.
    a. Varieties or stages.
    b. Pathological chemistry.
    c. Pathological physiology.
    d. Pathological anatomy.
        1. Gross.
            a. Size.
            b. Weight.
            c. Shape.
            d. Color.
            e. Surface.
            f. Capsule.
            g. Consistency.
            h. Cut section.
                1. Color.
                2. Surface
                3. Scrapings.

4. Morbid Anatomy—continued.
    d. Pathological anatomy—continued.
        2. Microscopical.
            a. Parenchyma.
            b. Stroma.
            c. Blood vessels.
            d. Lymphatics.
    e. Systems.
        1. Gastro-intestinal.
        2. Circulatory.
        3. Respiratory.
        4. Nervous.
        5. Genito-urinary.
            a. male.
            b. female.
        6. Osseous.
        7. Muscular.
        8. Lymphatic.
5. Symptoms.
    a. Varieties.
    b. Incubation.
    c. Onset.
    d. Common symptoms.
    e. Cardinal symptoms.
    f. General appearance.
    g. Physical examination.
        1. Inspection.
        2. Palpation.
        3. Percussion.
        4. Auscultation.
        5. Mensuration.
    h. Gastro-intestinal.
    i. Circulatory.
    j. Nervous.
    k. Special.
        a. eye.
        b. ear.

5. Symptoms—continued.
    l. Genito-urinary.
    m. Osseous.
    n. Muscular.
    o. Lymphatic.
    p. Respiratory.
    q. Laboratory examination.
        1. Sputum.
        2. Blood.
            a. culture.
            b. smear.
            c. cellular examination.
        3. Stomach contents.
        4. Urine.
        5. Feces.
    r. Röntgen-ray examination.
6. Course and duration. Complications and sequelæ.
7. Termination.
    1. clinical.
    2. pathological.
8. Diagnosis.
    1. direct.
    2. differential.
9. Prognosis.
    1. recovery.
    2. duration.
    3. complication.
10. Treatment. (Indications for treatment.)
    1. Prophylaxis.
        a. public.
        b. private.
    2. Active treatment.
        a. Hygenic.
        b. Mechanical.
            1. Hydrotherapy.
            2. Massage.
            3. Electricity.

10. Treatment—continued.
    2. Active treatment—continued.
        c. Medicinal.
            1. Etiological.
            2. Pathological.
            3. Symptomatic.
            4. Complication.
        d. Surgical—indications for same.
        e. Actino-therapy—indications for same.

CHAPTER III.

DISTURBANCES OF MENSTRUATION.

Hygiene of menstruation. The cardinal principle of the hygiene of menstruation is painstaking cleanliness. The external genitalia and pubic hair should be cleansed of adherent blood three times a day during the menses, as otherwise the blood decomposes causing a repugnant odor. The menstrual discharge should be received on pads fastened to an abdominal belt, to avoid soiling of the thighs and underwear. Such pads may be made from gauze with a layer of sterile cotton enclosed and should be burned after use. A thorough cleansing bath should be taken after menstruation.

Severe bodily exertion should be avoided during menstruation, such as bicycling, horseback riding, tennis playing, dancing, running a sewing machine, prolonged walking and cohabitation. Excessive mental exertion should be postponed, at least in young girls.

Sometimes an interval pain, or false period, occurs in some women, occurring in the interval between two menstruations. It is accompanied by the same symptoms as exist at the time of the menses, without, however, the loss of blood. This occurrence is generally without any clinical importance, being probably due to rupture of a Graafian follicle, which usually transpires about ten to fourteen days prior to the commencement of the next period.

The disturbances of menstruation are *physiological,* or

normal; *primary,* or essential; and *secondary,* or symptomatic. The primary anomalies are due to functional derangements of the uterus, the ovaries, or both, and are characterized by an absence of palpable pathological changes or objective signs in the pelvic organs. The *secondary* disturbances of menstruation are symptoms of organic, genital or constitutional diseases and presuppose the existence of palpable diseased changes in the genital organs.

The disturbances or anomalies of menstruation are:

1. *Amenorrhea,* an absence of the menses; and *oligomenorrhea,* a considerable decrease in the amount of the menstrual flow.

2. *Menorrhagia,* an abnormal increase in the amount of the menstrual blood lost; and *metrorrhagia,* irregular uterine hemorrhages independent of the menstrual period.

3. *Dysmenorrhea* is an abnormally painful menstruation. The pain must be severe enough to cause invalidism of the person.

1. Amenorrhea and Oligomenorrhea.

Etiology. These menstrual anomalies are *physiological, essential* or *secondary.* Physiological amenorrhea exists during childhood, pregnancy, the lactation period (first five months) and the climacteric.

The essential amenorrheas occur without any apparent cause. Women simply cease to menstruate. The condition may be temporary or permanent.

Secondary amenorrhea is caused either by genital or constitutional diseases.

Genital conditions accompanied by an absence or scantiness of the menstrual flow are (1) defective development, (2) surgical removal of all or part of the organs and (3) destructive disease processes.

Among such pathognomonic conditions are infantile

organs, uterine atrophy, aplasia and hypoplasia of the uterus or ovaries, destruction of ovarian tissues by tumor formation, especially of the malignant type, destruction of the endometrium caused by severe streptococcic infection and the cheesy form of uterine tuberculosis, resulting in necrosis of the uterine mucosa and finally lactation atrophy of the uterus and ovaries. Constitutional diseases accompanied by amenorrhea are chlorosis, tuberculosis, diabetes mellitus, obesity, various psychoses, severe nephritis, exophthalmic goitre, myxedema, Addison's disease, acromegaly and so forth. Morphinism and some other chronic poisonings also cause amenorrhea.

Symptoms. In the majority of cases amenorrhea is symptomless. In a small number of cases, so-called *molestiæ* or *molimina menstruationis* occur, consisting of irritability, pains in the pelvis, headache, intestinal disturbances, contraction of the breasts and an increase in the secretion of the uterine mucous membrane. In isolated cases *vicarious menstruation,* or hemorrhages from other parts of the body appear, as existaxis, melena, hemoptysis.

The *treatment* consists in attention to the primary disease. If the amenorrhea is caused by a delayed general or genital development, the system must be built up by proper food, exercise, rest, sojourns in the country and general hygienic care.

If molestiæ menstruationis exist, the mucous membrane of the cervix should be scarified, especially at the time of the expected menstruation. Other therapeutic measures include Sitz baths, sounding of the uterus, Bier's hyperemic method and, lastly, the insertion of an intrauterine stem pessary to irritate the endometrium. This should be worn from six to eight months or longer. Such a procedure is often effectual in bringing about a menstrual flow where all other measures of treatment fail.

The medicinal treatment includes iron preparations and arsenic in anemia and chlorosis, thyroid extract (grs. iii. t. i. d.) in obesity, antithyroid serum of Möbius in exophthalmic goitre, oophorin and yohimbin in infantilism. Other remedies recommended are potassium permanganate (0.1 gm. three or four times a day), salipyrin (1.0 gm. t. i. d.), diuretin (1.0 gm. b. i. d.), eumenol (4.0 cc. t. i. d.), aloin. Marriage sometimes removes amenorrhea immediately and permanently.

2. Menorrhagia and Metrorrhagia.

Profuse menstrual discharge may be the symptom of either a genital disease, as tumor of the uterus, inflammatory adnexal disease, perimetritis, displacement, and endometritis postpartum et abortum, or of a constitutional disease, as endocarditis, nephritis, disturbances of the ductless glands, as hypothyroidism and so forth.

Essential menorrhagia and metrorrhagia are conditions in which the menstrual flow comes too frequently, lasts too long, or is profuse without the existence of a demonstrable pathological basis. If the irregularity of the flow increases then these hemorrhages represent either apparent or irregular hemorrhages or metrorrhagias. Therefore essential menorrhagias or metrorrhagias may be different symptoms of the same disease. They are probably caused by a hyperfunction of the ovary or corpus luteum, resulting in an increase in the amount of the ovarian internal secretion.

The *symptoms* of essential menorrhagia and metrorrhagia coincide with those of a secondary anemia which follow the continued loss of blood, and are an extreme lassitude, inability to work, headache, palpitation of the heart, dyspnea, backaches, functional neuroses, and pain, resembling labor pain, caused by the expulsion of clotted blood from the uterine cavity. Dull pains and aches in the pelvis, back, loins and lower extremities, due to a

hyperemia or serous infiltration of the pelvic organs, are also met with.

Etiology. We do not know anything positive about the causes of essential uterine hemorrhages. Abnormal functioning of the ovaries (internal secretion and ovulation) is the logical presupposition for the occurrence of essential hemorrhages. This explains the predilection during puberty and the climacteric, when ovarian activity begins and ceases in the human body. The action of the ovarian hormone or internal secretion is inhibitory as well as stimulating to the function of the other ductless glands, and other ductless glands either inhibit or increase ovarian activity. Under these conditions, certain local or general disturbances may be of importance as predisposing especially to hemorrhage, for instance neuroses, chlorosis, congestion in the pelvis from tight lacing, the long-continued sitting position, sedentary habits, deficient physical exercise resulting from muscular insufficiency or weakness, constipation, machine sewing and enteroptosis. They all influence the blood circulation unfavorably and thus interfere with ovarian and uterine activity.

Further sources of hyperemias are abnormal sexual relations, ungratified sexual desire, onanism and coitus interruptus. *Psychic influences,* as sudden fright during menstruation, mental depression and worries, also influence ovarian activity and thereby menstruation. The cessation or absence of ovarian secretion causes an increased blood pressure, and this explains the occurrence of essential hemorrhages at the climacteric.

Pathology. As essential hemorrhages of the uterus signify a functional disease of the genital organs, the latter, therefore, do not show any recognizable pathological changes. This obtains in the majority of cases.

The *diagnosis* must be made by an exclusion of any other disease, local as well as constitutional, which may

cause such hemorrhages. This is done by a general and special pelvic examination. Bimanual palpation of the pelvic organs must not be neglected. Such a case should never be treated unless a careful microscopical examination of the endometrium has been made, as otherwise a cancer, sarcoma or tuberculosis might be overlooked, which would be of the gravest consequence. Adnexal disease and para- and perimetritis do not exclude the possibility of another disease of the uterus. Old infections of the uterus are to be excluded by a careful anamnesis (history) and repeated bacteriological examinations of the uterine secretions. Corpus hemorrhage must be distinguished from cervix hemorrhage. The former has periodicity, while the latter is irregular and follows local mechanical irritations, such as coitus, vaginal irrigations, examinations and physical exertion. A diagnosis of essential menorrhagias should be made only when the palpatory and objective findings of the pelvic organs are normal.

Without exception the *treatment* of essential uterine hemorrhages must be preceded by an exact diagnosis. A diagnostic curettage should be performed in all cases to exclude the existence of malignancy.

The *prophylactic* treatment is based on the demands of the underlying cause, such as anemia, neurosis, constipation, unnatural sexual relations and so forth. It includes a correction of injudicious behavior during menstruation (as undue physical and mental exertions, dancing, tennis playing, cold baths, becoming drenched, coitus, constipation continuing from early youth and sedentary habits). However, lukewarm bathing and moderate exercises are not injurious. The body must be developed and strengthened. A change of climate to a warmer region and higher altitude is often beneficial. Brine and iron baths are indicated, while mud-baths are contra-indicated. Douches of cold water on the lower abdomen

and the thighs, cold Sitz-baths, and cold water sprays are useful.

The *curative treatment* is either medicinal, mechanical, surgical or radiological. The *medicinal treatment* comprises ergot (1.0 cc 4 hrly.), hydrastis (0.5 cc 4 hrly.), stypticin (0.1 2 hrly.), pituitrin or pituglandol (1 cc of a 1:1000 solution hyperdermically), salpyrin (1 gm.), calcium chloride (1 gm. 4 hrly.), horse and human serum (10 to 20 cc), extract of the thyroid gland (0.2 t. i. d.)

By *mechanical* treatment an attempt should be made to immediately arrest a hemorrhage and to prevent the occurrence of future ones. One vaginal douche of ice-cold water, or a firm tamponing of the vagina, with absolute rest in bed, and an ice coil to the abdomen, may bring about a cessation of the bleeding. In the inter-menstrual period hot vaginal douches of one to two liters of water at 45 to 48 degrees C. (113 to 116 degrees F.) may be given every second or third day. To avoid scalding of the vulva and perineum vaseline should be freely applied to these parts, or a hot-water douche point used. Such douches have often been beneficial to ameliorate the menstrual flow.

If all these means prove of no avail, *surgical* procedures should be resorted to. These consist in a thorough curettage (10 to 20 percent. curative results in endometritis) followed by cauterization of the uterine cavity with liquor of iron chlorid, solution of zinc chloride (10 to 20 percent.), or tincture of iodine. Should this treatment fail, the uterine mucous membrane may be destroyed by the use of chlor-zinc paste or atmocausis (a vaporization with live steam at 106 to 125 degrees C. (222 to 257 degrees F.) for one-half to two minutes).

Radiological treatment consists in the use of massive x-rays (about fifteen to twenty erythem doses) until amenorrhea or oligomenorrhea are brought about, or the insertion of 25 to 50 milligrams of radium element into

the uterine cavity for about 10 to 20 hours. The radium must be well filtered to exclude the alpha and beta rays. The usual amount given is 500 to 1000 milligram hours. Radium therapy has rendered obsolete such mutilating operations as hysterotomy and hysterectomy. Radiological treatment results in a total cessation of menstruation. Hence it should be used only in persons in whom a premature or artificial climax is desirable.

3. Dysmenorrhea.

Painful menstruation is either a symptom of a genital disease, that is, a secondary dysmenorrhea, or a disease in which pathological changes cannot be found, that is, essential or primary. The pain in the pelvis must be so violent that the patient is incapacitated from work and rendered ill. Genital diseases that may cause dysmenorrhea are adnexal tumors and inflammations, submucous myomata, peri- and parametritis, bladder and bowel diseases.

Essential dysmenorrhea—symptoms and course. Pain is the only symptom. It is unbearable and labor-like, at times, but rarely, continuous, burning or boring. It is located in the pelvis from whence it radiates to the sides, legs and the small of the back. Vomiting, nausea, constipation or diarrhea, headaches, strangury, are often associated with dysmenorrhea.

Essential dysmenorrhea rarely occurs with the first menstruation. It develops gradually. However, it may appear suddenly, especially after a severe psychic shock or an injudicious exposure. Sexual intercourse tends to favorably influence dysmenorrhea, but still more favorable is the first labor. Multiparæ may acquire the disease. Changes in social conditions of life and of residence may have a favorable or unfavorable influence on menstruation.

The *etiology* is unknown. Predisposing factors are, (1) sensitive or over-sensitive nervous system, (2) delayed development of the genital organs, (3) mechanical obstruction to the outflow and (4) reflexly referred conditions.

(1) The uterine contractions present during each menstruation are felt as an unbearable pain by over-sensitive patients, and is like labor-pains.

(2) A hypoplastic or infantile uterus senses the pain as an over-distension, the unyielding external peritoneal and fibrous layers of the uterus contributing to an increase of this impression. This pain is felt as a dull discomfort, commencing a long time before menstruation, to disappear with the commencement of the flow.

(3) Another, and by far the larger, class comprise the mechanical dysmenorrheas. The pain is intermittent and colicky and ceases when the flow is fully established. In these women the uterine wall is rigid; the isthmus narrow. The premenstrual congestion of the mucous membrane almost or entirely closes the internal os. Severe uterine contractions are required to dilate the cervical canal and thus permit the blood contained within the uterine cavity to escape. In such patients the endometrium, and especially the internal os, is very sensitive. If a sound be introduced these patients almost bend double with pain and exclaim "this is my pain." In other words the internal os is hypersensitive.

Other causes of painful menstruation are the formation of blood clots in the cavity of the uterus the result of profuse menstruation, the expulsion of such clots being accompanied by pain. Hypersensitiveness of the sacro-uterine ligaments and ovaries also may cause dysmenorrhea.

(4) Conditions which reflexly cause dysmenorrhea are usually of nasal origin. They are relieved by cocainizing the lower turbinates and the tuberculum septi. A con-

nection between the nose and the genitalia exists due to phylogenetic reasons. Certain odors may lead to sexual excitation on the approach of male or female. Irritation of the nasal mucous membrane may cause uterine contractions even in narcosis.

Inflammatory diseases in the pelvis may leave behind a certain over-sensitiveness of the organs which may cause dysmenorrheic pains.

Pathological changes do not exist, though we may find a uterine hypoplasia or an anteflexio uteri with stenosis of the internal os. However, these conditions are not specific for essential dysmenorrhea.

The diagnosis can only be made by excluding diseases of the uterus and adnexa, the appendix, colon, bladder and ureters.

Treatment. Hypersensitiveness of the nervous system is corrected by building up the whole body through physical exercise, baths, and internal remedies.

Attention should first be directed to the *nasal mucous membrane.* Ten to twenty percent. solutions of novocain should be applied to the nasal mucosa. If relief is had, then cauterize the nasal mucous membrane. Seventy percent. of dysmenorrheas usually recover by this treatment.

The symptomatic treatment includes, amongst other remedies, aspirin (0.6 gm. 4 hrly.), antipyrin (0.6 gm. 4 hrly.), salpyrin (1.0 gm. 4 hrly.), diuretin (1.0 gm. 8 hrly.) and lastly, codein (0.03 gm. once) or morphin (0.015 gm. once) which are administered for the relief of the pain. Curative remedies are ergot (0.6 cc. 4 hrly.), hydrastis (0.3 cc. 4 hrly.), viburnum (fl. extr., 15 to 20 drops, t. i. d.). In mechanical dysmenorrhea stenosis of the internal os is removed by dilation and the insertion of an intrauterine pessary (the metranoikter). In hypoplasia uteri the development of the uterus is stimulated by intrauterine stem pessaries, Sitz-baths, hot vaginal

douches, and ovarian extract. Finally marriage may remove dysmenorrhea permanently, and especially the first labor. This fact should induce us to desist from any mutilating operation.

CHAPTER IV.

MALFORMATIONS OF THE VULVA, VAGINA AND UTERUS.

Malformations of the female genitals are congenital or acquired. The congenital causes are (1) a faulty anlage, (2) an arrest of development, (3) diseases of the embryo. The acquired deformities result from (1) inflammations, ulcer and gangrene of the genitals, (2) trauma sustained during operations and labor and (3) exanthematous diseases invading the genital organs. We may divide the malformations into (1) deformities of the external genitals, (2) aplasias, (3) atresias and (4) atrophy of the uterus.

Deformities of the External Genitals.

The rectum may terminate in the urogenital sinus either beneath or above the hymen, an *anus præternaturalis vestibularis.*

Imperfect closure of the primitive urethra toward the vagina or an absence of the posterior wall of the urethra is termed *feminine hypospadias.*

An imperfect closure of the urethra towards the clitoris, with an absence of the anterior wall of the urethra, is known as a *feminine epispadias.*

The presence of separate male and female sex glands in the same individual is termed *hermaphrodism.* True hermaphrodism has never been observed in the human race. However, *ovotestis* has. This is a state in which

typical testicular and typical ovarian tissues are present in the same sex gland.

Should the sex glands be either masculine or feminine and the external genitals have the partial or total characteristics of the opposite sex we speak of a *pseudo-hermaphrodism.* A feminine pseudo-hermaphrodism is a condition in which the sex glands are female and the external genitals have a more or less male type, and a masculine pseudo-hermaphrodism signifies the possession of male sex glands and the external genitalia are more or less of a feminine character. Pseudo-hermaphroditismus feminis is, indeed, rare. The external genitals in pseudo-hermaphroditismus masculinus resemble a rudimentary-like penis or hypertrophic clitoris, the urethra terminates beneath the clitoris, the scrotal halves do not coalesce, but resemble the large labia and often a depression, similar to a vagina, is observed.

Such individuals are raised as girls, may marry and pass through life as women, unless the appearance of secondary sex characteristics, habitus, beard, voice, and sexual manners create a doubt as to the proper sex.

Aplasia.

The aplasias are subdivided into (1) an absence and rudimentary development of Müller's ducts, (2) a rudimentary or partial development of one Müller duct with a complete development of the other duct. To these must be added (3), an incomplete fusion of Müller's ducts.

1. *A total absence of Müller's ducts* is very rare. The external genital organs are usually also incompletely developed. The ovaries may remain undeveloped.

If Müller's ducts remain rudimentary, the internal genital organs are palpated as bands and nodules running transversely across the true pelvis. They may or may not connect with the vagina. The latter is either of an infantile size and shape or forms a blind sac.

Symptoms. Women having such aplasias and rudimentary organs are usually hypoplastic and remain child-like. If the ovaries are absent or undeveloped the patient is sexless.

Should the ovaries mature then characteristic disturbances appear at puberty. They occur at regular or irregular intervals, depending on the periodicity of ovulation, and consist of menstrual molimen and vicarious menstruation.

The *diagnosis* is rendered by inspection and bimanual palpation through the vagina or rectum. The most important landmark to be located is the place of emergence of the round ligaments from Müller's ducts.

Treatment of the deformity is valueless. A vagina may be constructed if such a patient should marry and does not possess an organ for copulation. The best method of forming a vagina consists in making a canal between the bladder and rectum. This is done partially through a perineal incision, and partially by way of a laparotomy incision through the Douglas pouch. A portion of the ileum, about 30 cm. long, is separated from the bowel. It should have a sufficiently long mesentery and be so located that it may be drawn by a silk stitch attached at the middle of the loop, through the newly-made opening down to the perineal skin without undue traction. The resected portions of the ileum are united by a side-to-side anastomosis, and the abdominal incision may then be closed. An incision about 2 inches long is made in the middle of the transplanted piece of ileum and the edges carefully stitched to the introitus vagina with interrupted catgut sutures. The only objection to such a vagina is its abundant secretion, which will diminish by putting the patient on a diet free from albumin, but rich in fat.

2. *One of the Müller ducts may remain rudimentary and the other mature.* The developed portion communicates with the outer world, menstruates, serves its pur-

pose of cohabitation and may even form a womb for a fecundated ovum.

The rudimentary half may be only a band-like structure or be developed in certain parts. It may or may not coalesce with the other duct.

Should the developed portion menstruate, and not be fused with the normal half, the catamenial fluid will be retained. If the fluid collects in an atretic vagina it forms a hematocolpos, within the uterus a hematometra, and within the tubes a hematosalpinx.

Should an infection occur within this fluid a pyocolpos, pyometra or pyosalpinx results. If the bacteria perish in such a sac and the fluid becomes sterile then a hydrocolpos, hydrometra or hydrosalpinx remains.

Depending upon the amount of retained fluid and the pressure the latter causes within the occluded organ the sac may rupture. A rupture usually occurs towards the peritoneal cavity. Peritonitis follows or death may ensue from shock or infection.

The *diagnosis* is made from the history, the symptoms and signs. If a mass is found present on bimanual examination, if the patient complains of a periodic aggravation and an increase of pressure symptoms, and if the developed half can be plainly palpated the possibility of such a malformation should be considered.

The *prognosis* is good if the condition is recognized early and subjected to the proper treatment.

The *treatment* is surgical and consists in opening the sac and removing the contents.

3. *Müller's ducts may not coalesce* and we may find two separate genital canals; they may be partially fused and we then observe two uterine halves, *uterus duplex separatus,* or a cervix and two corporeal halves, *uterus bicornis unicollis;* or the uterus is of a normal form and the cavity is either entirely or partially divided by a membranous septum, *uterus septus or subseptus;* or the cer-

vical canal is traversed by a longitudinal septum, *uterus biformis.*

The *diagnosis* is made by inspection and palpation and by the insertion of two sounds. The sounds can be brought in contact where a septum does not exist.

Treatment is not necessary.

Atresia.

An atresia is a closure of the genital canal at any place in its course. It is most frequently found in the hymen and the vagina, rarely in the uterus and very seldom in the tube. At times several atresias are found present.

According to its location we have hymenal, vaginal, uterine and tubal atresias. The uterine atresias are divided into those of the external os, the cervical canal and the internal os.

Etiology. Atresias are either congenital or acquired. The congenital form originates from developmental disturbances and fetal inflammations. The acquired atresias result from genital inflammations, ulcerations and gangrene, exanthematous diseases, which are accompanied by an enanthem in the genital canal, surgical operations, neglect of uniting mucous membrane to mucous membrane, for instance, in amputations of the cervix, cauterizations and so forth, and, finally, trauma during labor.

Symptoms appear with the beginning of menstrual life. The menstrual fluid is retained. The patient complains of menstrual molimen or molestiæ occurring at regular intervals. The amount of retained menstrual fluid increases with each menstruation and finally causes pressure pain. The retained blood shows characteristic changes. The corpuscles shrink, the blood becomes gelatinous and tenacious and has a dark-brown or black color. The fluid may escape into the peritoneal cavity, setting up a peritoneal irritation, which is characterized by pyrexia,

pain, nausea and vomiting, or a hematocele may form, which usually undergoes absorption and resolution.

The *diagnosis* is made from the history of the case, the periodical menstrual molestiæ, the gradual increase of the pressure pain and the bimanual findings.

The *prognosis* is good if an early diagnosis is made and the atresia removed.

The *treatment* consists of a removal of the atretic membrane.

Atrophy of the Uterus.

Atrophy of the uterus is either congenital or acquired. The causes of congenital atrophy are arrest of development and the organ remains either infantile *(infantile uterus)* or it assumes the form and shape of an adult uterus, but remains very small *(hypoplastic uterus)*. The causes of acquired uterine atrophy are (1) physiological processes, as senile atrophy, transient lactation atrophy, or puerperal hyper-involution; (2) premature senile atrophy following castration operations; (3) lactation atrophy following protracted nursing; (4) severe septic infections of the uterus and ovaries, resulting in a complete destruction of either the endometrial or ovarian tissue; (5) general constitutional diseases, as chlorosis, anemia, lues, diabetes mellitus, nephritis, suppurations, morphinism, adiposity, severe psychoses, and sojourn in tropical climates.

The acquired uterine atrophy is either *concentric,* in which the organ becomes symmetrically smaller and shortened to about four or five centimeters, or *eccentric,* in which the uterine walls become thin, soft, flabby without the uterus decreasing in length.

The *infantile uterus* retains the characteristic size and form of the uterus of an infant, is about four to five centimeters in length and the proportion of the length of the body to that of the cervix is as one to three.

The genital organs remain infantile and general infantilism of the patient is not uncommon. The breasts are small and flat, the growth of hair in the axilla and external genitalia is scant, the deposit of fat in the mons veneris and labia majora is small, the perineal body remains low, the hips do not widen and so on. Menstruation is always absent.

The *diagnosis* is made by measuring the body and cervix with the sound.

Treatment is of no avail.

The *hypoplastic uterus* has the form of the uterus of a pubescent woman, its development in size and length, however, is retarded. It is most frequently found in otherwise poorly-developed individuals, especially those having a congenital hypoplasia of the heart and vascular system. The cause of hypoplasia of the genital organs, as well as of the vascular system, is a disturbance in the physiological equilibrium of the ductless gland system. The exact pathology is as yet unknown.

Symptoms. Menstruation may be present or absent, the possibility of the occurrence of conception is, therefore, apparent. Sterility is, however, present in the great majority of cases. If menstruation is absent, periodical menstrual molestiæ or molimen may occur, accompanied by a profuse leucorrheal discharge, but without any admixture of blood.

The *diagnosis* is obtained by bimanual palpation and sounding.

Treatment is hardly ever effectual in marked cases. One may try a general building up of the body by nourishing foods, especially milk, physical exercise, baths, and iron and arsenic treatment. We may use, locally, scarification of the cervix, irritation of the endometrium by the sound or the introduction of an intra-uterine stem pessary. Sitz baths, hot vaginal douches up to 40 to 50 degrees C. are advisable. Eventually we may recommend

a currettage to attempt to render the mucosa more favorable for the implantation of a fecundated ovum. Pregnancy actually has occurred after such a treatment.

Atrophia Uteri. The microscopic findings are the same as those of a senile uterus. The muscle tissue is wanting.

Symptoms. These are amenorrhea, menstrual molimen and sterility. In some cases the molestiæ menstruationis may be absent.

The *diagnosis* is made by palpation and sounding.

Treatment depends on the cause. If the cause cannot be removed therapy is entirely unnecessary. In lactation atrophy the patient must wean the infant. In tropical atrophy she must be removed to the temperate zones. Adiposity must be corrected by diet, exercise and thyroid treatment. Other diseases must be removed, as anemia, chlorosis, diabetes, hyperthyroidism and so forth.

The local treatment attempts to produce a congestion of the uterus and thus induce the organ to menstruate. It consists of scarification of the cervix, Bier's hyperemic treatment, daily warm vaginal douches at 40 degrees C. and the insertion into the uterus of a stem pessary. Medicinally potassium permanganati (0.5 to 1.0 gm. q. i. d.), Yohimbin (0.01 gm. q. i. d.), corpus lutem extract (0.2 gm. t. i. d.) are valuable.

The state of the development of body and general health must be improved by proper diet, sojourns in the country and at the seashore, wholesome, nourishing food, horseback riding, extended walking and training in a gymnasium under the direction of a trained teacher. Lawntennis, golf and so forth are invaluable to these patients.

CHAPTER V.

DISPLACEMENTS OF THE UTERUS AND VAGINA.

The *typical position of the uterus* is the anteverted, in which the fundus lies posterior to the upper border of the symphysis with the cervix in the interspinous plane. The fundus, in a cranio-caudal direction, should not rise above the inlet of the pelvis, and the portio should not descend below the interspinous line. The uterus should not deviate towards the right or left of the median line. The position is passively changeable by distension of the bladder or rectum, or both. Any anteverted position within the described limits is, however, normal.

A *permanent displacement of the uterus* is pathological. Limitation or arrest of the normal movability of the uterus is the chief characteristic of its displacement.

The Different Positions of the Uterus.

We distinguish the following different *positions* of the uterus: I. *Versions:* (a) Around a transverse axis, retroversion. (b) Around an antero-posterior axis, latero-version. Sinistro-version, if the fundus turns towards the left with the cervix towards the right. Dextro-version, if the fundus turns to the right with the cervix to the left. (c) Around a longitudinal axis, torsion or rotation. Dextro-torsion, if the right side of the uterus turns forward. Sinistro-torsion, if the left side of the uterus turns forward.

II. *Displacements of the entire uterus:* (a) Antero-posterior. 1. Ante-position, if the uterus, *in toto,* is displaced forwards. 2. Retro-position if it is displaced backwards. (b) Lateral. 1. Dextro-lateral if the uterus is displaced to the right. 2. Sinistro-lateral if displaced to the left. (c) Cranio-caudal. 1. Elevation, if the uterus ascends above the pelvic entrance. 2. Descent, if it descends down to the vulva, and 3. Prolapse, if the uterus descends below the vulva.

The different changes in the *form* of the uterus are:

I. *Flexions:* (a) In the normal ante-flexion the body forms an angle of about 90 degrees with the cervix. Beyond that and forming an acute angle the ante-flexion is pathological. (b) If the cervix and body form an angle which opens posteriorly it is called a retro-flexion. (c) If the body is flexed laterally towards the cervix we have a latero-flexion, and we further distinguish dextro- and sinistro-flexions.

II. *Elongation* of the uterus is a lengthening of the organ due to pressure or traction.

III. *Axial rotation* is a turning of the corpus uteri towards the cervix around a vertical axis.

IV. *Inversion* of the uterus is the turning inside out thereof.

Displacements and deformities of the uterus may be primary, or essential, if they occur without any other pelvic pathology. They are then a disease entity.

Changes in the position and form of the uterus may be a symptom of diseases of other pelvic organs. For instance, a tumor in the anterior wall of the uterus may cause a backward displacement of the organ. Or a perimetritis may cause adhesions between the uterus and the pelvic walls, and thus displace the uterus. Again, the patient may have a perimetritis, the displacement of the uterus being only a concomitant symptom. Or an in-

terstitial myometritis may render the uterus rigid and cause a pathological ante-flexion. The patient is then not suffering from an ante-flexion but from a myometritis, the ante-flexion being a result of the inflammation of the myometrium.

Etiology of Uterine Displacements.

The weight of the uterus proper is carried by the suspensory apparatus, which is formed by the peritoneum, ligaments, connective and muscle tissue. As their length almost corresponds to the distance between the points of insertion in the uterus and the bony pelvic walls, they are also able to fix the uterus to a certain degree. However, they could not permanently resist increased intra-abdominal pressure, if the latter did not become arrested by the resistance of the muscular and bony abdominal walls. The pelvic floor is the inferior portion of the abdominal wall. The abdominal pressure forces the pelvic organs towards the walls, and a further displacement of the organs is prevented by the resistance offered by the walls. This force is exerted by the levator ani muscle and the muscles of the uro-genital diaphragm, which constitute the supporting apparatus of the pelvic organs. As long as these muscles remain intact and functionate normally prolapse cannot occur.

Retroversio-flexio Uteri.

Etiology. This condition arises when the vesico-uterine pouch becomes filled with intestinal loops. The pouch is empty under normal conditions. Intra-abdominal pressure acts on the anterior uterine wall and if for any reason the contents of the recto-uterine pouch become displaced upwards the body of the uterus is forced into the hollow of the sacrum. A distended bladder and rectum may force the small intestines out of the cavum Douglasii (Douglas cul-de-sac). As soon as the

rectum is emptied the uterus is forced into the hollow of the sacrum.

The predisposing causes which enable the intestines to enter the vesico-uterine pouch are a relaxation of the suspensory apparatus of the uterus, anomalies of development, tumor formation and inflammations in the recto-uterine pouch.

A relaxation of the suspension apparatus results from subinvolution during the puerperium, constitutional diseases, as chlorosis, tuberculosis, wasting diseases, infantilism, asthenia and masturbation. Anomaly of development is a congenital shortness of the anterior vaginal wall. The pressure of tumors on the anterior wall, or traction on the posterior wall, may mechanically displace the uterus from its anterior position. Inflammatory processes in the recto-uterine pouch may cause retro-displacement by fixing the uterus in Douglas' pouch by means of peritoneal adhesions (perimetritis posterior).

Pathology. Different changes in the topography of the pelvic organs are found according to the degree of the displacement. The posterior pouch may be free of intestines, and the anterior filled with bowel. The posterior wall of the uterus may rest upon the rectum or posterior pelvic wall. The tubes and ovaries may be correspondingly displaced, the round and sacro-uterine ligaments becoming straightened.

The form of the uterus may be changed. The body may be larger as a result of congestion. The angle between the body and the cervix may not open anteriorly, but posteriorly. The curve of the uterus may depend on its bed or support. If the corpus lies lower than the cervix a retroflexion results. The upper vaginal walls become folded if a descent occurs. Adhesions, the result of peritoneal inflammations, may cause the uterus to be fixed.

Symptoms. A retroversio-flexio uteri may be symptomless, in which case it does not possess any clinical

importance and, therefore, does not require treatment. When evident the symptoms are either mechanical or reflex. They differ according as to whether they occur, 1, in nulliparæ, also termed congenital, or 2, during pregnancy, and 3, during, or following, the puerperium.

1. Congenital retro-flexion of the uterus. Mechanical symptoms are pressure on the rectum and backache with constipation, bladder irritation and a feeling of fullness and heaviness in the pelvis.

Congestion of the uterus causes a hyperplastic endometritis with dysmenorrhea, menorrhagia, leucorrhea and sterility or lessened fertility.

Reflex disturbances are digestive disorders, hemicrania, neurasthenia and so forth.

The *diagnosis* is made by a bimanual examination which reveals that the cervix points anteriorly, the body being located posteriorly to the cervix. If the uterine body is found beneath a plane through the cervix a retro-flexion exists. Mobility and immobility are to be also noted.

The *differential diagnosis* takes into consideration retro-flexion, tumors in the posterior uterine wall, and adnexal tumors adherent to the posterior uterine wall. (See tables on differential diagnosis.)

2. Retro-flexio Uteri Gravidi (Retro-flexion of the Pregnant Womb). If pregnancy occurs in a retro-flexed uterus, an incarceration of the organ within the true pelvis may take place. This accident is rare. The symptoms of incarceration are a sense of fullness in the pelvis and an obstruction of the bladder and rectum, causing ischuria and finally ischuria paradoxa and obstinate constipation. Such urinary disturbances in a pregnant woman should always invite suspicion to the possibility of a displacement of the uterus.

Diagnosis. The pelvis is filled with the enlarged and retro-flexed uterus. The bladder and bowels are dis-

tended. Catheterization and flushing of the bowels will clear up conditions.

3. Puerperal Retro-flexion. The majority of the retro-displacements of the uterus occur after a full-term labor or an interrupted pregnancy (abortion).

(a) If it occurs during early puerperium, that is, within the first fourteen days after delivery, then rectal tenesmus, urinary disturbances, severe hemorrhages and backache appear. On examination the still enlarged uterus is found no longer above the pelvic brim, but rather it occupies the true pelvis. Usually an exploration of the uterine cavity reveals remnants of placental tissue.

(b) Retro-flexion of the uterus during the late puerperium (third to sixth week) is characterized by a bloody vaginal discharge, pain in the pelvis, and, if placental tissue remains, bad-smelling secretions and the expulsion of tissue débris. The uterus then is large and soft, i. e., subinvoluted.

(c) The symptoms of a post-puerperal retro-flexion are a history that the patient did not regain her former health after the last labor, and has dysuria and frequent urination, constipation, dysmenorrhea, and menorrhagia, sterility, leucorrhea and a sense of weight in the lower portions of the pelvis. These pelvic symptoms are generally accompanied by constitutional symptoms, such as loss of weight and strength, nervousness, anorexia, digestive disturbances, headaches and sacraches (pain in the sacral region).

The *prognosis* of all varieties of retro-versio-flexio is good if the organ may be replaced in its normal position.

Treatment is indicated. 1. If symptoms exist which are directly traceable to the displacement. 2. If an endometritis complicates the displacement. 3. In operations for descent, or prolapse, of the uterus and vagina.

The treatment has a twofold purpose. 1. To replace

the uterus in a normal position, and 2, to maintain it in this position.

Acute and subacute inflammations absolutely *contra-indicate* any treatment, because an exacerbation of the inflammation, or a rupture of existing pus sacs, might occur. Adhesions or agglutinations which fix the uterus should be separated before a replacement is attempted. This may be done by massaging according to B. S. Schultze's method.

The *reposition* of the uterus may be performed by a *bloodless* or by a *surgical* procedure. In the former the uterus is replaced by means of bimanual reposition and then held in the normal position by a Hodge or Thomas pessary. The pessary must be large enough to retain the uterus in ante-version. It should not cause the patient any pain by pressure. The lower transverse bar of the pessary should be wide enough so the instrument rests on the pubo-coccygeal portion of the levator ani muscle, otherwise it may slip out on bearing-down efforts of the patient. The patient should return to the physician every four weeks for a re-examination. She should also be directed to use a daily cleansing vaginal douche and to return immediately to the physician if any untoward symptoms appear, such as pressure-pain, profuse leucorrhea, odor. Otherwise the pessary may be worn for six to eight months, when it is removed. The patient then should be objectively, or at least subjectively, cured. About ninety per cent. of such displacements and deformities recover under this treatment, so that only a small percentage remains to be subjected to surgical treatment.

The indications for operative treatment are, 1, where pessary treatment fails; 2, if the patient cannot wear a pessary because of relaxed vaginal outlet, or refuses to submit to the treatment (virgins); 3, where existing adhesions cannot be separated, and 4, if we must subject

the patient to an operation for other pathological conditions (descent and prolapse).

The different methods of operation may be classified as follows: 1. The fixation operations. (a) Ventro-fixation and (b) vagino-fixation. 2. Shortening of the round ligaments. (a) Intra-abdominal duplications (Webster-Schmitz). (b) Ventro-suspension (Doléris-Gilliam). (c) Vaginal duplications (Wertheim). (d) Vesico-uterine fixation through the vagina. (e) Shortening through the inguinal canal (Alexander-Adams).

The treatment of an *incarcerated pregnant uterus* demands vesical catheterization, flushing of the rectum and replacement of the uterus by bimanual manipulations, eventually under anesthesia. Should this treatment not bring about the desired results a reposition must be made through an abdominal incision. Pregnancy may then proceed to full term.

The treatment of *retro-flexions* of the uterus *occurring* during the *early* or *late puerperium* consists in a careful curettage of the endometrium, removal of any retained parts of the ovum, replacement of the uterus into anteversion and maintaining it in this position by a pessary.

Ante-flexio Uteri.

An ante-flexion of the uterus becomes pathological when the angle between the body and cervix is less than 90 degrees, or is acute.

Etiology. A tumor located anterior to the uterus may pull, or a tumor posterior to the organ may press, on the body and cause an ante-flexion. The change in form, however, is a congenital defect in the majority of cases and is usually associated with a hypoplasia and stenosis of the cervix. The cervix may also become displaced, its axis pointing anteriorly towards the symphysis pubis.

Anterior perimetritis between the bladder and uterus

and chronic myometritis resulting in a rigidity of the uterus are not unusual causative factors.

Symptoms. A pathological ante-flexion may exist without symptoms. The symptoms, if existing, are dysmenorrhea and sterility, both of which are the result of a mechanical obstruction to the outflow of the menstrual discharge produced by the flexion.

The diagnosis is easy and is made by a bimanual examination.

The *treatment* consists in a dilatation of the cervical canal, and Dudley's plastic operation for ante-flexion. (See *American Journal of Obstetrics*, 1891, No. 2, Vol. 24.)

Descent and Prolapse of the Uterus and Vagina.

Etiology. Any disease factor disturbing the supporting structures of the pelvic organs (levator ani muscle and urogenital diaphragm), creates a disposition to a descent and prolapse of the genital organs. The ever-existing intra-abdominal pressure exerted upon the pelvic organs is not then neutralized by an intact pelvic outlet, hence a displacement downwards of the pelvic organs follows.

The disease factors causing a relaxation of the pelvic diaphragm are, 1, direct injuries of the muscles of the pelvic floor, and 2, diseases of the nerves supplying these muscles.

Injuries or *defects* of the muscles of the pelvic floor may result from (a) *trauma* during the process of labor, producing lacerations of the muscles of the pelvic floor or a detachment of these muscles from the bony pelvis. (b) *Infections,* either strepto- staphylo- or gonococci invading the muscles of this region and resulting in necrosis or fatty degeneration of the muscle cells (phlegmon). (c) Severe or long-continued *pressure* on the pelvic muscles by impacted pelvic tumors, terminating in a

sloughing and loss of tissue. (d) Chronic, long-continued local or *general illnesses* as anemia, tuberculosis, wasting diseases, causing atrophy and degeneration. (e) *Advanced age* and accompanying senile atrophy.

Diseases of the nerves result from (a) *pressure* and *contusions* of nerves supplying the pelvic muscles (the pudendal nerves and the cervical ganglion). (b) *Inflammations* of the nerves, 1, of the epineurium (sheath of nerves), and 2, of the perineurium (sheath of nerve trunks) resulting in atrophy of the muscles through loss of innervation, or by extension of inflammatory processes from the nerves to the muscles.

Pathology. The uterus is suspended within the pelvis and held in ante-flexion by the uterine ligaments, which are condensation zones of the endo-pelvic fascia and contain an admixture of muscle fibres derived from the myometrium. They alone, however, cannot resist undue intra-abdominal pressure which continually acts from above and tends to displace the pelvic organs downward.

Intra-abdominal pressure is neutralized by the resistance offered by the bony and muscular walls of the pelvis (the pelvic bones and the pelvic muscular diaphragm), provided they are in a perfectly normal condition, the muscles not having been injured or enervated. The muscles chiefly concerned are the levatores ani and the transversi perinei. The pubo-coccygeal portions of the levatores ani are pierced anteriorly by the urethra and the vagina and posteriorly by the rectum. The genital cleft is, under normal conditions, closed by the intact pubo-coccygeal portion of the levatores ani. If, however, an injury involves the levators, a divergence of the pubo-coccygeal portion of the levatores ani is established, which forms the natural predisposition to the occurrence of a prolapse. For the urogenital cleft may be aptly compared to an abdominal ring, such as the femoral. Atrophy or injury of the muscles lining the ring causes

dilation of the latter and hernia formation. The internal abdominal organs lying near such a relaxed opening are then easily forced out of the abdominal cavity. According to the degree of divergence of the levatores ani the uterus, bladder, rectum, and vagina may be forced out of their normal confines, i. e., through the pelvic diaphragm, and a prolapse of these structures therefore results. A uterus in a normal ante-verted position cannot descend. In order for a prolapse to take place the uterus must first become retro-verted so that its axis is in alignment with the axis of the vagina. Then the intra-abdominal pressure will cause the uterus to pass through the pelvic floor by descending through the vagina. As a senile uterus is smaller than a normal one, and as the cervix is barely one-third as wide as the fundus, the fact of the great frequency of prolapse of these uteri is explained, the uterus acting as a wedge.

The following facts demonstrate the value of muscle as supporting structures in contra-distinction to fascia: 1. The amount of muscle tissue in the pelvic floor decidedly surpasses that of fascia. 2. In the abdominal wall muscle tissue, and not the fascia, is the resisting tissue. A ventral hernia forms in the linea alba if diastasis of the recti muscles occurs. 3. A surgeon would not think of uniting a celiotomy incision without interposing a layer of muscle between the peritoneum and superficial fascia.

Symptoms. The symptoms are those of a retro-versio-flexio uteri, plus a feeling of openness of the vagina and a sensation as if the abdominal organs wanted to fall out. Interference in the functions of the bladder, rectum and vagina, and dragging pains in the pelvis, are always present. The necessity of manually replacing the prolapsed organs to permit an evacuation of the bladder and rectum cause a depressed state of mind. These disturb-

ances disappear when the patient assumes the recumbent position.

The general symptoms are loss of appetite, weight and strength, sexual invalidism, loss of earning capacity and of the enjoyment of life.

Objective signs. On inspection one ascertains the protrusion of the genitalia, with the corresponding diverticuli of the bladder and rectum; also epidermification of the exposed mucous membranes and the formation of decubital ulcers.

Varieties of descent and prolapse: 1. Descent of the lower half of anterior wall with a diverticulum of the posterior bladder wall (cystocele). 2. Descent of the upper half of the anterior vaginal wall with enterocele. 3. Descent of the upper half of the posterior wall with enterocele. 4. Descent of the lower half of the posterior vaginal wall with rectocele. 5. Prolapse of the lateral vaginal walls. 6. Descent and prolapse of the retroverted uterus. An ante-verted uterus will descend or prolapse only if Douglas's pouch is abnormally deep and the levatores ani markedly displaced downward, causing a descent of the ante-verted uterus downward and backward.

The *diagnosis* is made by inspection. The patient should be instructed to bear down when the prolapse is completely developed. She is then told to contract the levator ani muscles, when the degree of divergence of their pubo-coccygeal portion can be determined by palpation.

The *prognosis* depends on the degree of divergence of the pubo-coccygeal portions of the levator ani muscles. 1. If the genital cleft after contraction of the muscles narrows down to the width of one or one and a half fingers, the prognosis as to a permanent cure is absolutely good. 2. If it remains wide enough during contraction to admit two fingers, the prognosis should be

guarded. 3. If the cleft remains wider than three fingers, the prognosis is absolutely bad. According, therefore, to the width of the genital cleft, the prolapse is spoken of as of the first, second or third degree.

The *treatment* is prophylactic, palliative, and curative or surgical.

Prophylaxis. We must attempt to avoid forceps and surgical deliveries, guard the intactness of the hiatus (genital cleft) during the passage of the fetus and immediately repair injuries to the muscles of the pelvic diaphragm occurring during labor. The muscles of the pelvic floor should be stimulated to resume their normal form and elasticity by recommending an early rising, gymnastic exercises of the lower extremities and the abdominal muscles, external douches of alternate hot and cold water, Sitz baths and astringent vaginal douches during the puerperium.

The *palliative treatment* should be followed in all cases in which operations are contra-indicated on account of age, general physical defects, or constitutional diseases. It consists in the use of pessaries, as the soft-rubber, the Gehrung, or the ball pessary. The instrument must be large enough to prevent its escape through the abnormally wide genital cleft.

Surgical treatment attempts to repair the suspension apparatus, as well as the pelvic floor, in one sitting. The proper procedures depend on the results of the examination, which determines the position of the uterus, the width of the genital cleft, and the conditions of the uterus, vagina and perineal body. 1. In all cases of the first degree of divergence, we should perform an anterior and posterior colporrhaphy, Hegar's perineorrhaphy, curettage of the endometrium and amputation of the cervix, if indicated, and also ventro- or vagino-fixation if the uterus is retro-flexed. If the uterus is in anteversion it does not require any attention during the

operation, as an ante-verted uterus cannot descend. 2. In cases of the second degree, we should, instead of the perineorrhaphy, make an isolated transversi perinei profundi and levatores ani suture and correct any uterine pathology. 3. In prolapse of the third degree, which is practically incurable, we narrow the lumen of the vaginal canal and genital cleft by a levator ani muscle suture and perform a hysteropexy, according to Kocher's method. This may give tolerable relief for many years.

Lacerations of the Perineum. Perineorrhaphy.

Etiology consists of: (a) *Injuries from within,* as during the delivery of the child at full term, or the removal of a tumor through the vagina. Labor, however, is by far the most common cause. (b) *Injuries from without,* as falls, blows, forcible sexual intercourse, and so forth.

Pathology. Perineal lacerations are either *incomplete,* when they involve the vaginal mucosa, the perineal skin and the muscles of the perineal body; or *complete,* when the rectal mucous membrane is torn in addition. The tear usually follows the raphé perinei, extending upward either into the left or right sulcus, or both. As a rule it extends higher on the side where the occiput presents. The collumnæ rugarum are hardly ever injured. The direction of the tear is up and down. A gaping of the vaginal entrance occurs and the tear, if left to itself, heals in a transverse direction. A disposition to the formation of a rectocele follows. The anterior and posterior walls are exposed to view. If the support of the pelvic diaphragm is destroyed, depending on simultaneous injury of the levator ani muscles and the urogenital diaphragm, a prolapse of the vaginal walls occurs with a corresponding cystocele or rectocele.

If the laceration involves the rectal mucosa a complete perineal laceration results, and as a rule its direction

follows that of the vaginal tear. The anal opening gapes and an ectropion of the rectal mucous membrane, with a prolapse of the rectum, results. The stumps of the anal sphincter retract decidedly, thus causing a dimple formation bilaterally.

Symptoms. The immediate result of a perineal laceration is hemorrhage, which is seldom severe. The parts, if left to themselves, heal by cicatrization. The remote results are irritation of the exposed parts of the vagina and rectum. A sensitiveness of the parts, increased on walking and exercising, is at times complained of. If the laceration is complete, incontinence of feces results. Other symptoms attributable to the rectocele and cystocele are complained of as a feeling of openness of the vagina and a constant tendency of the parts to prolapse.

The *prognosis* is favorable if the defect can be immediately repaired. If the laceration is corrected afterward by a perineorrhaphy then the result is never as good as following an immediate repair.

Treatment. The immediate repair may be executed as follows: The parts are rendered surgically clean. The patient is preferably given an anesthetic. The injured tissues are united with catgut, layer by layer. (a) The rectal mucous membrane by a continuous mattress suture, which should not include the epithelial surface covering of the rectum. (b) The vaginal mucous membrane should next be coaptated by a continuous catgut stitch. (c) The structures of the perineal body are then brought in apposition by interrupted catgut stitches. (d) Lastly, the skin is closed by a continuous horse-hair, or continuous catgut, interrupted stitch.

The operation for an old laceration varies according as to whether it is complete or incomplete. Various methods have been advised. 1. Emmet's operation: The scar tissue is outlined by an incision and removed. The denuded surfaces are approximated by transverse sutures.

2. Tait's flap-splitting perineorrhaphy: A transverse incision is made and the vaginal mucous membrane dissected up for a variable distance. The structures of the perineal body are united anatomically by transverse continuous catgut sutures. 3. Levator ani muscle suture method: A transverse incision is made. The vaginal mucous membrane is dissected off the underlying structures until the levator ani muscles are exposed. If the sphincter ani and rectal mucous membranes have been torn then the stumps of the sphincter muscle are also to be dissected out. The sutures to approximate the sphincter ani are introduced from one side, and pick up the rectal mucosa like a purse-string, without perforating into the lumen of the rectum and then pick up the opposite stump. Three such sutures are to be used. When these are tied the sphincter and also the rectal mucous membrane are in apposition. The remaining structures of the perineal body are coaptated by interrupted catgut stitches, layer by layer; 1, the levator ani muscles; 2, the transverse perinei muscles; 3, the superficial fascia, and 4, the vaginal mucous membrane and skin.

Inversio Uteri.

Etiology. An inversion can only occur when the uterine wall is relaxed and flabby and the cervical canal and uterine cavity are dilated. Pressure from within or traction from without may then invert the organ. Pressure from within results from performing Credé's method of delivering the placenta. Traction from without is caused by pulling on the umbilical cord. These conditions always occur during the third, or placental, stage of labor. A non-puerperal inversion may be caused by pulling on a pedunculated tumor, which is extruded into the vagina.

There are three degrees of inversion: (a) depression,

(b) invagination (incomplete inversion), and (c) complete inversion.

Pathology. Inversion is *incomplete* if the fundus is either depressed into the cavum uteri (depression), or invaginates and appears at the external os uteri (invagination). It is *complete* if the fundus and corpus are completely inverted and lie in the vagina. Complete inversion may, at times, be associated with a prolapse. The inverted uterus has the form of a bag. Its outer surface is formed by the uterine mucous membrane, the inner lining by the serous covering of the uterus. Should the inversion exist for some time, a replacement of the cylindrical surface epithelium by squamous epithelium may occur, which leads to an epidermification. The formation of ulcers on the mucous surface is possible. If the constriction of the cervical ring is marked, circulatory disturbances, as edema and increase in the size of the uterus, appear. In a puerperal inversion, the place of insertion of the placenta may be seen. An involution of the inverted uterus may also take place if the displacement is not corrected.

Symptoms. Inversion of the uterus occurs suddenly and with threatening symptoms during the puerperium, while its development is rather slow and gradual in an extra-puerperal inversion. The first symptom is a profuse hemorrhage, which may cause shock or even a fatal termination. At times the hemorrhage is small in amount, but frequently repeated. The inverted portion of the womb may become gangrenous, especially if the cervical ring is firm and contracted. The symptoms of a chronic inversion are irregular hemorrhages, due to erosions of the endometrium, profuse discharges and irregular and profuse menstruation. Tenesmus, a bearing-down pain, and backache are frequent.

The *diagnosis* is made by establishing the fact that the corpus uteri does not lie above the pear-shaped mass in

the vagina, and that the inversion funnel can be palpated through the abdominal walls.

The *prognosis* is always bad. Sometimes a spontaneous recession of the uterus may occur, or the uterus may be replaced without difficulty and remains in the replaced position. This naturally renders the prognosis favorable. The prognosis is bad on account of the danger of hemorrhage, infection, and rupture of the uterus during attempts at replacement.

Treatment. Prophylactic. Be careful during the delivery of the placenta or of polypoid pedunculated tumors. Do not pull on them. *Curative.* An inverted uterus should be replaced immediately at the time of the accident. The sooner the inversion is discovered and treated, the easier the replacement. In cases of inversion where the placenta is still attached to the fundus, a replacement should be attempted with the placenta in situ by preference. The reposition is executed either manually or by surgical operation. The manual reposition is accomplished after anesthetizing the patient as follows, an even pressure is made on the uterus through the vagina and counterpressure on the inversion ring through the anterior abdominal wall. The cervix may have to be dilated. If manual reposition fails surgical procedure should be instituted. Surgical reposition is performed by making an incision in the posterior vaginal fornix. A finger is passed into the funnel of the inverted uterus and the posterior uterine wall incised in its entire length. The uterus is then replaced and the incisions in the posterior wall of the uterus and vaginal fornix closed.

CHAPTER VI.

INFLAMMATIONS OF THE VULVA, VAGINA AND UTERUS.

Inflammatory diseases of the female genital organs form a most important group and comprise a large percentage of the diseases peculiar to women.

Inflammation in any portion of an organ affects either the parenchyma (epithelial and glandular tissue), causing a parenchymatous or *glandular inflammation;* or the connective tissue, terminating in an *interstitial inflammation.* The inflammation may be acute or chronic. Glandular inflammation is characterized by an hyperplasia, hypertrophy and, if the result of an infection, by a round and plasma-cell infiltration; while the interstitial inflammation causes pressure atrophy of epithelial tissues due to an increase in the connective tissue. Hypertrophic inflammation is characterized by a more profuse secretion, leucorrhea and pyorrhea, and in addition menorrhagia if the internal generative organs are also involved. If atrophic inflammation is present it is evidenced by a decrease, and finally cessation, of the glandular secretion and oligo- or amenorrhea with an involvement of the uterus and adnexa.

In the majority of cases (90 per cent.) the inflammation depends on an infection with bacteria (infections), and in the minority is caused by chronic circulatory disturbances (catarrhal inflammations).

Infections appear chiefly in three varieties: the gonor-

rheal, septic and tuberculous. Other but rare infections are the syphilitic and the chancroidal.

Gonorrheal infection is caused by the gonococcus, the septic by the strepto- and staphylococci, more rarely the colon bacillus, typhoid bacillus, pneumococcus, actinomyces, diphtheria bacillus and various other unidentified bacteria, possibly those responsible for measles, scarlet fever, variola and so forth. Tuberculous infection is due to the tubercle bacillus, syphilitic to the spirochetæ pallidæ and chancroidal to Ducrey's bacillus.

The nature of the infection depends upon incidents of sexual life (cohabitation, labor, and the puerperium), surgical instrumentation (operations, examination, sounding) and from primary foci of infection in distant parts of the body (intestinal infections, pulmonary tuberculosis).

The routes of infection are three: 1, ascending; 2, descending, and 3, hematogenous.

In *ascending* infections the pathogenic organism enters the genital canal from without and migrates from below upwards. The ascending infection is exclusively attributable to the gonococci, strepto- and staphylococci and at times the tubercle bacilli and rarer the colon and typhoid bacilli. It is brought about, 1, by an antiperistalsis; 2, by wandering leucocytes, and 3, by a backing up of the secretions.

In *descending* infection the pathogenic bacteria enter the genital organs from within. The micro-organisms migrate through the abdominal organs to the pelvic organs if peritoneal adhesions exist between them (appendiceal inflammations, adherent intestines). The micro-organisms concerned in this mode of infection are the bacillus coli, bacillus tuberculosis, bacillus typhosis and actinomyces.

The *hematogenous* infection carries the pathogenic micro-organism from distant foci through the blood

stream to the pelvic organs. The bacteria concerned in this mode of infection are the tubercle bacillus, the pneumococcus and, rarely, the typhoid bacillus.

Infections from without are also termed exogenous, those from within endogenous.

The *chronic* circulatory disturbances are due to active, or arterial, and passive, or venous, congestion brought about, 1. By pelvic circulatory disturbances caused (a) by trauma and the subsequent formation of scar tissue, (b) by displacement of the pelvic organs, (c) by tumor formations, (d) by torsion and strangulation of the pelvic organs, (e) by hernias of the pelvic organs and (f) by a long-continued coprostasis or fecal impaction. 2. By constitutional diseases accompanied by circulatory disturbances, as (a) cardiac, (b) renal and (c) hepatic diseases.

Various physiological processes in the female facilitate the entrance of pathogenic bacteria into the pelvic organs. These danger periods are menstruation, pregnancy, labor and the puerperium. A high percentage of infections occurs at these times.

Recognition of the infective organism is of considerable importance as an aid to prognosis, diagnosis, and treatment. This is particularly so in the acute inflammatory conditions, where the administration of an appropriate antitoxic serum or autogenous or stock vaccine may make a great difference in the chances of the patient's recovery. In chronic cases the identification of the organism may be impossible, however, it is not of as much importance as in the acute infection.

The lesions to which these infections give rise may be classified anatomically according to the part affected, and have been termed vulvitis, vaginitis or colpitis, cervicitis, endometritis, myometritis, salpingitis, oophoritis, perimetritis, or pelvic peritonitis and parametritis, or pelvic cellulitis.

The majority of infections terminate in a complete cure, with restitutio ad integrum. If retention of the purulent secretion, abscess formation or cicatricial contraction occur, the parenchyma may become destroyed, with a corresponding loss of function (amenorrhea, sterility).

Inflammations of the Vulva.

1. Acute Inflammations.

Acute septic vulvitis is not as frequent as septic infections of other parts of the genital canal. The predisposing causes are inflammations or disintegrating tumors of the vagina and uterus, vesico-vaginal and recto-vaginal fistulæ, infections of the urethra, diabetes mellitus and lack of cleanliness. The direct or primary cause is a loss of continuity of the surface epithelium which is necessary for the entrance of the infectious agent. The epithelial surface of the vulva usually possesses a sufficient resistance to prevent the entrance of the germs.

The *symptoms* are *redness, swelling, secretion* and *pain.* Reddening is either diffuse or localized in spots. The swelling may be very extensive and usually is most prominent at the hymen and the small labia. The secretion is mucous, muco-purulent or purulent and of a whitish-yellow, often greenish, color, at times watery, again thick and tenacious. Pain is experienced as a fullness and sensation of heat in the labia, or a burning on urination, and is increased on palpation or upon walking. When the acute stage subsides a pruritus follows which is at times very tormenting. The objective signs correspond to the subjective symptoms.

The actual causative factor and the underlying primary disease must be sought for to enable one to properly treat the vulvitis.

Acute gonorrheal vulvitis constitutes about seventy per cent. of all cases. It is usually primary in nulliparæ and secondary in multiparæ, in whom the cervical gonorrhea is primary.

The accompanying infection of the urethra, of the paraurethral or Skene's and Bartholin's glands is clinically characteristic of a gonorrheal vulvitis. These structures are hard to the touch, sensitive on palpation, and show macular red discolorations around the external openings of the ducts (maculæ gonorrheica).

The secretion is profuse, purulent, white or greenish. Redness, swelling, discharge and pain are more prominent than in the other forms of vulvitis.

Bartholinitis gonorrheica is either unilateral or bilateral. It often leads to the formation of an abscess having the size of a plum or egg. The temperature remains until the abscess is either evacuated or spontaneously ruptures. The abscess is usually located in the posterior third of the labia majora.

Condylomata acuminata, or venereal warts, often accompany gonorrheal vulvitis. They are papillary proliferations, or warts, due to a proliferation of the papillary bodies of the skin; they form on the small and large labia and may involve the entire perineum. At times they coalesce, leading to the production of cauliflower-like masses. They may also be caused by other acrid discharges.

Infantile gonorrheal vulvitis may be caused by sexual indulgence and contact with infected sponges, cloths, bath-tubs and so forth.

The *symptoms* are redness, swelling, secretion and pain. They are much more aggravated than in the adult and the beginning is usually accompanied by general malaise, pyrexia, and a swelling of the inguinal lymph nodes.

The *diagnosis* of all the inflammations of the vulva is based on a microscopical examination of the discharge.

Gonorrheal infections are diagnosed by staining a smear preparation for half a minute in an alcoholic solution of methylene blue. The gonococcus is the only coccus decolorized by Gram's method; above all it is found *within the pus cells* and can be grown only on human serum.

Lues vulvæ (hard chancre) must be diagnosed by the finding of the spirochetæ. It occurs oftenest on the frenulum, clitoris and inner surfaces of the labia minora. The initial sclerosis, edema of the labia and finally ulcer formation are characteristic. Multiple primary lesions of the vulva are the rule, while the primary lesion of the cervix is always solitary.

Ulcus molle vulvæ (soft chancre) is more frequently found at the frenulum and is always multiple, has sharp-cut, distinct edges and gives rise to a profuse discharge and a painful swelling of the inguinal lymph glands, which tend to suppurate. The diagnosis should be confirmed by finding Ducrey's bacillus.

Infectious exanthematous vulvitis is caused by acute infectious diseases as diphtheria, scarlet fever, measles, small-pox, pneumonia, erysipelas and actinomycosis. Dysentery, typhoid fever and cholera also may invade the vulva by direct contact from the rectum. These infections very frequently terminate in a hymenal or vaginal atresia.

The skin diseases of the vulva. Eczema intertrigo is the most frequent and attacks the large labia and the genital and femoral sulci. It results from secretions, uncleanliness, especially in the hot seasons of the year, and leads to soreness, redness, and itching.

Seborrhea is due to an increased activity of the sebaceous glands, causing accumulations of a whitish substance on the labia.

Acne pustulosa and furuncles may result from scratching.

The *diagnosis* of all the forms of acute vulvitides is based on bacteriological and microscopical findings. The course is usually rapid and the symptoms disappear early under proper treatment. Frequently, however, a subacute or chronic vulvitis follows, especially the acute gonorrheal form.

The *prognosis* depends on the etiologic factor.

Prophylaxis includes thorough cleanliness, avoidance of unnecessary handling of the genitals, and paying special attention to these organs during the existence of infectious diseases.

The *treatment* consists in the removal of the cause. If this is impossible then it must be symptomatic. Cleanse the parts thoroughly and keep them clean by warm Sitz baths. Absolute rest in bed is important. Boric acid dressings, lead acetate dressings, applications of zinc ointment, xeroform and so forth are indicated. The treatment of gonorrheal vulvitis is the same plus the application of silver salt solutions (protargol 1 per cent., silver nitrate 2 per cent.).

Bartholinitis requires hot boric acid dressings. If an abscess forms it should be incised. If it ruptures, drain and keep the parts clean.

The treatment of venereal warts consists in cauterization with chromic acid, or the actual cautery, and dusting with pulv. frondum sabinæ and calomel āā.

Vulvo-vaginitis infantum should be treated by local cleanliness, irrigations with iodine or potassium permanganate (1:5000) solutions and subcutaneous injections of autogenous vaccine. ℞ Potassi iodi 5.0, tr. iodini 25.0, aquæ q. s. ad 175.0. M. et signa: Use one tablespoonful in three quarts of warm water as a douche.

Lues requires excision of the primary ulcer, followed by cauterization, and the intravenous administration of salvarsan.

Ulcus molle demands excision or daily cauterization of

the ulcer with phenol, full strength, followed by moist dressings of liquor plumbi subacetatis dilutus.

In acne and furuncle apply tincture of iodin, or an ointment composed of ichthyol, 1.0, sulph. precip. 10.0, zinc ointment 25.0, or, if necessary, the lesions may be incised.

Eczema and seborrhea should be treated by borax washings and applications of dilute resorcin-alcohol solutions of one-half per cent. strength.

2. Chronic Inflammations of the Vulva.

The causes are neglected acute vulvitis, and persistence of the primary disease causing the acute form. The gonorrheal variety tends frequently to become chronic. Chronic irritations of the vulva may cause a hyperemia (masturbation, incomplete coitus, inability of emmissio penis and anti-conceptional measures).

The symptoms are itching, burning, intermittent secretions and the formation of reddish, discolored maculæ.

Treatment: Remove the cause. Use cool Sitz baths. Enforce strict cleanliness.

Pruritus vulvæ is a continued or intermittent itching, occasionally accompanied by a burning sensation.

The *pathological* changes are an hypertrophy of the connective tissue of the papillary bodies, a sub-epithelial, round-cell infiltration, an increase in the size and number of sebaceous glands and, finally, a localized parakeratitis, the result of scratching and rubbing.

Etiology. Often an external irritant is present, such as a discharge from the uterus due to cervical catarrh or cancer, pathological urine, masturbation or incomplete coitus, diabetes mellitus, bladder or kidney stones, icterus, obstinate constipation and intestinal parasites.

At times a cause for the pruritus cannot be found. It

then seems to be a neurosis, when it is known as an *essential pruritus vulvæ.*

The *symptoms* are itching, especially at night under the warmth of bed clothing, insomnia, general irritability and finally melancholia.

The *signs* consist of a lead-gray discoloration of the vulva. The latter is flabby and leather-like, the result of the irresistible scratching.

Treatment. Remove the cause if possible. Locally use Sitz baths and applications of three to five per cent. phenol-solutions, two or three per cent. silver nitrate solutions, dusting powders, one per cent. menthol solutions, tincture of iodine. Finally x-ray or radium may be resorted to, which sometimes effect a cure when all other means fail. The hypodermic injection of autogenous vaccine from the vaginal and vulvar secretions has been highly extolled by some authors.

Kraurosis vulvæ is a rare disease characterized by a cicatricial contraction of the entire vulva, a narrowing of the vaginal orifice, an absence of the glandular elements of the skin, loss of hair and an asbestos-like, white, shining appearance of the integument.

Pathology. It is the final stage of chronic inflammatory vulvitis, characterized by an absence of the papillæ, a cicatricial contraction of the connective tissue and a complete destruction of the elastic fibres of the skin.

Symptoms. Mild cases are quite without symptoms. Advanced cases complain of a painful itching, especially during urination, along with a sensation of contraction, chiefly in the hymen.

Treatment. If the disease causes symptoms excision of the affected parts is indicated.

Elephantiasis vulvæ is a marked increase in the size of the vulva, especially the labia majora, and is characterized by an edema, sclerosis of the connective tissue and a lymph stasis.

It is caused by an obstruction of the lymph vessels, due to an inflammatory vulvitis, or to an excision of the inguinal lymph nodes.

The treatment consists in an excision of the vulva.

Ulcus rodens vulvæ is a rare ulcer formation of the vulva, usually located in the navicular fossa and characterized by a progressive destruction and invasion of the contiguous tissues of the vagina, and leading to the formation of urinary and fecal fistulæ.

The *symptoms* correspond to the extent of the ulceration.

The *differential diagnosis* must be made between rodent ulcer and lues, cancer and tuberculosis. The cause and pathology of rodent ulcers are not known.

The *treatment* is surgical and consists of an excision of the entire mass, which usually means the entire vulva.

Inflammations of the Vagina.

General etiology. The predisposing causes of vaginitis are 1. injuries during coitus, labor, gynecological examination and instrumentation, 2. insertion of foreign bodies into the vagina, usually pessaries. The *exciting causes* are infections, either ascending from the urethra, anus and vulva, or descending from the peritoneum, tubes and uterus. Hematogenous infection of the vagina is rare.

Acute colpitis or vaginitis invades the mucosa and submucous connective tissue.

Causes. The inflammation is the result of an invasion of the vagina by gono-, strepto- and staphylococci. The natural protection of the vagina consists in, 1, the acid reaction of the vaginal secretion, which delays the growth of pathological bacteria until the latter turn acid fast, and 2, the character of the squamous epithelium, which offers resistance to the entrance of bacteria. However, a change in the acidity of the vaginal secretion, continued

irritation leading to a destruction of surface epithelium, and injuries and loss of continuity finally create a portal of entrance for the germs.

Pathology. The diseased changes occur in the submucosa and are characterized by a hyperemia, an edematous enlargement of the epithelium and a round-cell infiltration either localized or diffuse. Finally the squamous surface epithelium may partake in this process.

The *symptoms* are discharge and pain. The discharge is opaque, serous and granular with an admixture of cell elements and creamy if pus cells are numerous. Pain is present only in the acute stages and is of a severe burning character, with an unbearable sensation of fullness and heat, a soreness in the deep parts of the pelvis and at times vesical and rectal tenesmus.

The signs consist in diffuse changes in young sufferers, and localized granular changes in the old. The mucosa is covered with numerous red spots, the size of a pinhead, so that it looks as if afflicted with flea bites. The papules bleed easily and are readily palpable. The loss of surface epithelium may lead to an agglutination of opposing surfaces of the mucosa and an atresia vaginalis.

The prognosis as to a cure is not favorable, as exacerbations are frequent, due to a continuation of the primary cause.

The *treatment* consists in absolute rest in bed, interdiction of coitus, and removal of the source of the infection. We may apply locally glycerin tampons: in gonorrhea tampons with glycerine 100, camphor 1.0, alumnol 2.0 and boric acid 10.0, or use the dry treatment with bolus alba or silicat. Tincture of iodin should be applied to the ulcerations.

If the patient cannot go to the physician's office vaginal irrigations with potassium permanganate, 2 percent. boric acid or aluminum acetate solutions may be ordered, and in gonorrheal infections 2 per cent. solutions of ar-

gonin or protargol, or vaginal suppositories of alum or tannic acid (1.0 or 0.5 gm. in 2 to 3 gm. cocoa butter).

Colpitis emphysematosa occurs usually during pregnancy, to disappear during the puerperium. The only symptom is a discharge.

Signs. On palpation soft nodules the size of a split pea, which disappear on pressure, are felt. On inspection transparent white elevations, which emit a gas on being punctured, are seen.

Etiology. It is caused by a non-malignant gas bacillus. The prognosis is good. Treatment is unnecessary.

Acute exanthematous colpitis. This form of vaginitis occurs during measles, scarlet fever, diphtheria, smallpox, typhoid and cholera and leads frequently to an atresia vaginalis. It is very rare.

The *symptoms* and *signs* are severe pains in the region of the vagina, contractions of the muscles of the pelvic floor, swelling of the submucosa, profuse purulent secretion and loss of surface epithelium with the subsequent formation of ulcers. Agglutinations of the vaginal walls or contraction of the vagina may follow.

Treatment. Remove the cause. Make daily applications of tincture of iodin. Vaginal irrigations and the insertion of xeroform (deodorized iodoform) gauze strips, to prevent stenosis and atresia, are essential.

Chronic mechanical colpitis is caused by masturbation, excessive and abnormal coitus, badly-fitting and long-worn pessaries, and prolapsus. A concurrent infection may render the condition worse. The result of mechanical irritation is the formation of ulcers, decubitus or pressure gangrene, which usually remain localized.

The *prognosis* is favorable if the cause is removed.

Treatment. Remove the cause and dust the ulcer with an antiseptic powder until healing ensues.

Ulcers of the vagina often offer difficulties for differential diagnosis. They may be primary syphilitic

chancres, ordinary traumatic ulcers, infectious phlegmonous ulcers, cancerous ulcers and, finally, soft chancres, which latter, however, are much more rare in the vagina than in the vulva.

The *diagnosis* should be a bacterial and histologic one. Bacterially the demonstration of the presence of the spirochetæ pallidæ, tubercle bacilli, Ducrey's bacilli is made. Cancer, tubercle, necrosis and ordinary infections are to be differentiated by the microscope.

Ulcers may lead to extensive scar formations, with stenosis and atresia of the vagina.

Paracolpitis. This is phlegmon or inflammation of the connective tissue surrounding the vaginal canal.

Etiology. Paracolpitis is usually the result of a trauma sustained during labor, and injuries of the vaginal walls by violence from without which offer an entrance to highly virulent pathogenic bacteria, usually the streptococci. It may involve the entire pelvic region and be accompanied by extensive destruction and sloughing of tissues.

The *prognosis* depends on the kind and virulency of the invading bacteria and the general resistance of the patient. Finally atresias may form, due to cicatrization following the sloughing of tissues.

The *treatment* consists of rest, antiseptic irrigations, incision of abcesses and general constitutional and symptomatic attention. Atresia is to be prevented by the insertion of gauze strips into the vagina.

Inflammations of the Uterus.

Uterine inflammations invade (a) the mucosa, either the cervical, causing endocervicitis or the corporeal, causing endometritis and (b) the muscle or wall, either of the cervix, causing cervical hypertrophy, or myocervicitis, or of the corpus, causing myometritis. Such inflammations may be acute or chronic, infectious or non-

infectious. These inflammations also include those pathological changes brought about by circulatory disturbances, such as venous and arterial congestions. They are really not of an inflammatory character. German writers have proposed to term these "simple catarrhal changes, or hyperplasias." Let us adopt this classification, which is chiefly based on the investigations of Hitschmann, Adler and Hans Schroeder.

The infectious form usually appears in four varieties: 1. Saprophytic, caused by saprophytic and putrefactive organisms, 2. Septic, chiefly caused by the strepto- and staphylococci, 3. Gonorrheal, 4. Tuberculous. To these may be added, 5. Luetic infections, due to spirochetæ pallidæ.

A saprophytic infection is a bacterial intoxication due to the action of the products of bacterial growth. The saprophytes may become lodged in wounds, set up irritation there and without themselves gaining entrance into the tissues, give off toxins for absorption. They only infect the mucous membrane and do not penetrate the walls of the uterus. These micro-organisms maintain their growth only on necrotic tissue-débris retained in the uterus, and succumb rapidly after the removal of the débris due to the reactive power of the host.

Septic infections always gain entrance through a defect in the surface continuity of the endometrium. Either they remain limited to the mucous membrane or invade the myometrium and even reach the perimetrium. Such an extension depends on the virulence and the number of the bacteria, as well as on the resisting power of the tissues and the host. Pregnancy, labor, the puerperium, menstruation and trauma during gynecological examination and operations form the means of entry for these bacteria.

The *gonoccocus* is essentially a mucous membrane parasite. Its deposition on a mucous membrane suffices for

the beginning of this infection. The infection usually remains confined to the mucous membrane. It is only exceptionally that deep structures become involved.

The tubercle bacillus causes either an ascending or primary infection, a descending or secondary infection, or a hematogenous infection from a distant primary focus. Deposition of the bacillus on the mucous membrane under favorable circumstances forms tubercle.

The *luetic* infection is either secondary or tertiary, and depends on a primary lesion elsewhere, usually of the vulva or cervix. Loss of continuity of the surface epithelium is necessary for the production of a primary lesion.

The clinical picture of all these inflammations of the uterus is the same and therefore infections will be treated collectively, except the tuberculous. Tuberculosis will be considered in a special chapter while the reader is referred to books on syphilology for the study of the syphilitic affections.

Endocervicitis.

Inflammations of the cervix depend on the continuous contact of the latter with a pathological vaginal secretion. The internal uterine os forms a natural barrier to the extension of the inflammation to the corpus and therefore the cervix alone remains diseased.

Endocervicitis originates in the cervical mucous membrane and from there spreads to the walls of the cervix. The *direct causes* are either infection or chronic hyperemia. The *predisposing causes* are the incidents of sexual life, labor, the puerperium, surgical operations, gynecological examinations and the delivery of large intrauterine tumors through the cervical canal. The most frequent injury of the cervix is a laceration. If it becomes infected it does not heal by primary intention, but by granulation and scar formation. The result is a gaping

of the external uterine os and the lower cervical canal. An eversion of the cervical mucosa and ectropium of the cervical lips take place.

Pathology. The mucous membrane is hyperemic and edematous, its surface is knobby, uneven and deep-red. The knobby elevations, which are papillary, bleed easily. If a blocking of the glandular secretion due to occlusion of the gland ducts occurs, retention cysts form.

The knobby elevations are known as *papillary erosions,* the retention cysts as *follicular erosions* (mucous patches). The cervical mucous membrane becomes everted or the laceration exposes the cervical mucous membrane to the injuries of the vaginal secretions. Such a condition is known as an ectropium.

Microscopically a proliferation of the surface and glandular epithelium and connective tissues, a round and plasma cell infiltration and an increase in the number and size of the capillaries, are apparent. The demonstration of plasma cells renders the only proof of the existence of an infection in chronic inflammatory disease. Plasma cells are of lymphocytic origin. They are rounded or polygonal in shape with eccentric nucleus and basophyle cytoplasm, often exhibiting a clear space or zone at one side of, or around, the nucleus.

Papillary and follicular erosions encroach onto the vaginal mucous membrane and the line of demarcation between the vaginal squamous cell surface epithelium and the cervical cylindrical cell epithelium becomes irregular and indistinct. The vaginal mucosa encroaches on the cervical, or the cervical on the vaginal. If a cervical gland becomes closed by squamous epithelium, retention cysts, known as *ovula Nabothi,* are formed.

Symptoms. Acute endocervicitis is characterized by an increased *secretion* of a thin, serous, creamy or purulent nature. It causes a *burning sensation* if it comes in contact with the vulva. *Fever* occurs only in highly viru-

lent infections. *Chronic endocervicitis* causes a *profuse discharge* and a *dull ache* in the deeper parts of the pelvis. The secretion is abundant, of a purulent character, with a bad odor if the patient is uncleanly. There is a burning or itching sensation in the vulva. If eversion of the mucous membrane or erosions are present, a bloody admixture to the discharge is frequent, especially following coitus, instrumentation or examinations. The foregoing lesions and ovula Nabothi may cause a feeling of pressure and pain in the cervix.

A formation of cicatricial tissue around the external os may result in a stenosis. The cervical secretion becomes blocked and is retained, because the internal os prevents a backward escape of the secretion. The result is a forcible elongation of the cervix. If the inflammation finally subsides the elongation and stenosis of the cervix remain. The clinical importance of elongatio cervicis will be discussed in the section on cervical hypertrophy.

The *signs* of acute endocervicitis are *tumefaction* of the entire cervix, intense *reddening* and thickening of the cervical mucosa, and the escape of a profuse watery or creamy purulent *secretion* from the external os. *Papillary erosions* are recognized by the warty, knobby nodules on the surface of the cervical mucosa, they bleed easily on irritation. *Follicular erosions* are diagnosed by puncturing the retention cysts, when the mucous contents escape. *Ovula Nabothi* may be seen through the usually transparent squamous epithelial cell covering. If we puncture such cysts a purulent or watery fluid exudes.

A bacteriological examination of the discharge of an acute endocervicitis shows pathogenic bacteria, while such a demonstration is usually negative in the chronic form.

Treatment. Prophylaxis. The prevention of infec-

tions during surgical interference and gynecological examinations, abortions, labor, and the puerperium is most important and demands strictest asepsis.

Curative. The *acute inflammation* should be treated by rest in bed and cold compresses, or a Leiter coil, to the lower abdomen for the relief of pain. External douches may be used to ameliorate the burning of the vulva. As soon as the fever subsides we may use hot vaginal douches of solutions of salt or sodium bicarbonate. It is needless to state that abstinence from sexual intercourse is imperative and that local medication and instrumentation are to be condemned.

Chronic Endocervicitis. Erosions and eversions of the mucosa should be treated by scarifications, followed by the use of a Bier's cup and the insertion of a tampon. The treatment may be repeated every day until the erosions or hypertrophy disappear. The tampon should be removed after six to twelve hours. Vaginal douches of a normal saline solution should be used once daily. The amount of water employed should be three quarts. After four to six scarifications have been made, applications of a saturated alcoholic solution of picric acid, tincture of iodin, liquor of iron chloride, or iodized phenol may be of advantage. These applications may be followed by the insertion of tampons saturated in pure glycerin, or a 10 percent. glycerin solution of tannic acid, or a 10 percent. glycerin solution of ichthyol. If a cure does not occur with this treatment within four to six weeks the erosions should be excised.

Chronic cervicitis, the result of *lacerations,* should always be treated surgically. The proper operation relieves the ectropium of the cervical mucous membrane immediately (Emmet's trachelorrhaphy). Generalized endocervicitis usually demands a cervical amputation if it does not improve under medical treatment (circular or v-shaped excision and high or low amputation).

Endometritis.

Pathology. In acute recent cases gonorrheal, saprophytic and septic infections always cause a severe interstitial endometritis, i. e., 1. a marked proliferation of the interstitial tissue, 2. an extensive round and plasma cell infiltration, either diffuse or focally arranged and gradually decreasing in number from the surface to the deeper structures, and 3. a surface necrosis of the mucosa. The gonococcus is essentially a mucous membrane parasite and usually invades the endometrium. In saprophytic infections the dam of round cell infiltration is always confined to the mucosa. A deeper penetrating involvement is rather an exception. While in septic infections the round cell infiltration has a tendency to advance along the blood vessels to the muscle layer, and finally to the perimetrium. Often the blood and lymph vessels are clogged completely by the cocci.

If a *mixed* infection is present, and in the gonorrheal infections they always occur, the puscocci displace the gonococci and the septic tends to preponderate over the saprophytic infection.

A *saprophytic acute endometritis* tends to complete recovery. The material on which the saprophytes thrive is consumed entirely or removed, or the resisting powers of the organism which become readily activated, nullify its action and overpower it.

Acute gonorrheal and septic endometritis usually terminates in the *chronic form,* provided the bearer of the disease does not succumb to a severe general infection, i. e., septicemia or bacteriemia. On microscopic examination of the chronic form, areas of round and plasma cell infiltration are found scattered about in the hypertrophic mucosa. There is a serous infiltration of the connective tissue and a dilatation of the blood and lymph vessels. The glands may also partake in the pathological

changes, becoming hypertrophic and hyperplastic. It is usually impossible to find the primary causative disease factor.

This chronic inflammation may gradually lead to a complete recovery with a restitution of all normal functions, so that menstruation, pregnancy, labor and the puerperium may run their normal physiological course. If the chronic inflammatory process, however, continues then the connective tissue of the mucosa proliferates, the epithelial cellular elements atrophy and finally disappear entirely. The mucous glands become compressed by the increase of the connective tissue and succumb. Finally an atrophy of the connective tissue of the entire mucosa ensues. This atrophy cannot be distinguished from a senile atrophy occurring at the menopause.

The atrophic chronic endometritis runs a slow course, extending over years and finally passes imperceptibly into the climateric.

If the destruction and necrosis of the tissues, i. e., mucosa and upper muscularis, have been extensive and rapid, a premature climax occurs with a premature senile atrophy of the entire uterus.

An atresia of the internal os leads to a retention of the infected material and discharges within the uterine cavity and a pyometra results. The surface epithelium and glands disappear. This occurs especially in infections in elderly women. The purulent material is generally expelled by intermittent contractions of the uterus causing labor-like pains.

Rarer infections of the endometrium may be caused from typhoid, diphtheria, small-pox, scarlatina and so forth. They run the same course as septic endometritides and may also terminate in a premature climax.

In other cases of chronic endometritis a general hypertrophy and hyperplasia of the mucosa, especially of its glands, continues. This may be differentiated from the

glandular hypertrophy and hyperplasia of the premenstrual congestion by a round and plasma cell infiltration in places, the remnants of the former acute infectious process. There is usually an increased vascularity, the walls of the capillary vessels being somewhat thickened.

These changes result in an increased thickness of the mucous membrane and to the naked eye it looks more vascular and spongy. In some cases the thickening is so great as to take the form of velvety projections, very like those present in a carcinoma of the endometrium.

Hyperplastic Endometritis.

Chronic non-inflammatory or *catarrhal endometritides,* are caused by agencies inducing a chronic passive or active hyperemia. *Venous stasis* is brought about by retro-flexio uteri, myomata, ovarian and tubal tumors, chronic constipation, cardiac, hepatic and nephritic diseases and *arterial stasis* from masturbation, venereal excesses, interrupted coitus, defective hygiene or neglect during menses and so forth. All chronic inflammations which are preceded by a labor, abortion or the puerperium should be considered as infections unless otherwise proven.

Such chronic catarrhal endometritides are usually characterized by a profuse *leucorrhea* and *menorrhagia.* It is unquestionably true that chronic venous and arterial stasis induce an increased ovarian activity, i. e., an increase in the amount of ovarian hormones, which, in turn, leads to an increase in the duration of the menses or in the amount of menstrual fluid, or both.

Pathology. On macroscopic examination a thickening of the mucosa to one centimeter, and single or multiple mucous polypi, are seen.

Microscopically hyperplasia and hypertrophy of the glands are apparent, as shown by invagination of the gland ducts, pseudo-acinous arrangement of the glands,

with a corkscrew-like, or serrated, form on longitudinal section. The stroma is decreased. An increased amount of glandular secretion is the result. If the gland ducts should close from pressure retention cysts form. Such a condition has been termed *cystic glandular endometritis.* Hypertrophy and hyperplasia of the uterine glands are also met with in the mucosa during the period of premenstrual congestion. However, if they are found present during the post-menstrual period of reconstruction, they should be considered as pathological and are then designated hypertrophic catarrhal endometritides or hyperplasias.

Symptoms. Acute endometritis begins with *chills and fever.* The latter is mild and usually disappears rapidly. *Pain* accompanies the disease in the form of pressure and weight in the pelvis. There is vesical and rectal tenesmus or sensitiveness. A *profuse purulent secretion* is pathognomonic in this disease.

The *symptoms* of all forms of *chronic endometritis* are a *profuse discharge* with *menorrhagia* and, more rarely, *pain. Sterility* is the rule. The discharge is either serous or purulent, aqueous or creamy. It is present at all times. The menorrhagia is marked, leading often to metrorrhagias. The pain is dull and intermittent and felt in the depth of the pelvis. Exacerbations at the time of the menses are usual. Dysmenorrhea, however, is seldom observed.

General disturbances of health and earning capacity have been attributed to chronic endometritis. These are digestive disorders, anorexia, headaches and severe nervous symptoms, also secondary anemia and chronic ill health due to the severe losses of blood.

Signs. Acute endometritis is diagnosed by a bacterial examination. Bimanual palpation elicits an enlarged uterus, tender to pressure. Inspection reveals a profuse secretion at the external genitalia and cervix.

The signs of a *chronic endometritis* are very indistinct. Profuse leucorrhea is the only specific evidence. Only microscopical examination renders the diagnosis positive. Either the presence of plasma cells is then demonstrated, when the chronic inflammation was primarily caused by an infection, or hypertrophy and hyperplasia of the glandular elements is found during the period of post-menstrual reconstruction, when a chronic catarrhal hypertrophic endometritis or hyperplasia exists.

Bimanual palpation elicits the enlarged uterus, sensitive to pressure. Exploration of the uterine cavity with the sound reveals a marked sensitiveness and an irregularity of the surface of the mucosa. Examination of the secretion is negative.

The *differential diagnosis* is important and can be made only by the microscope, which enables a recognition of a tuberculous, luetic or cancerous disease from endometritis.

The *prognosis* is good in the acute form, but must be guarded in the chronic, on account of the tendency to recurrence after an apparent cure. Conception may take place in the severest forms, pregnancy going on undisturbed to full term, with labor and the puerperium normal; while in other, and even milder, forms, sterility may exist, or if conception occurs, pregnancy is interrupted, or the patient is continually ill, and the puerperium finds her with a renewed activity of the inflammatory process.

Treatment is prophylactic and curative. Prophylaxis demands prevention of infection. The treatment of *acute endometritis* consists of rest in bed, interdiction of sexual activities, instrumentation and examinations. The general nutrition should be kept up. Pain is combated with cold compresses to the lower abdomen. Profuse discharge demands external douches. After the acute stage has subsided sojourns in the country or mudbaths (Wau-

kesha) are beneficial. *Chronic endometritis* calls for an abrasio mucosæ, followed by a cauterization of the mucous membrane with tincture of iodin, liquor of the chlorid of iron, iodized phenol or phenol and alcohol. Packing the uterus with Beck's bismuth paste is also good. The tampon should be removed after seventy-two hours.

Electro-therapy in chronic endometritis is poor treatment. Atmocausis is dangerous. Actino-therapy is indicated if the medical treatment fails to cure.

Hysterectomy, finally, is justifiable for severe, chronic endometritis which resists the above relief and in which actino-therapy cannot be made use of, a state of affairs which certainly should not exist in an up-to-date gynecologic clinic.

Metritis.

1. Metritis acuta. *Etiology.* This condition is the result of an extension of an acute endometritis and is always infectious. The septic infection occurs during abortions, labor, the puerperium and trauma; and the gonorrheal from contact with gonorrheal pus. The invasion of the myometrium by the gonococcus is, however, rare.

Pathology. The infective organism invades the myometrium through the lymphatics. These vessels are filled with micro-organisms, which migrate into the connective tissue between the muscle fibres. A perivascular round and plasma cell infiltration forms. A thrombo-phlebitis of the capillaries is not unusual, especially at the site of the placental attachment. The infection may involve the entire myometrium and even spread to the perimetrium and pelvic abdomen.

Occasionally abscesses form in the uterine wall, which break down, leading to a sloughing of tissue. The ab-

scesses usually point into the uterine cavity, but may also rupture in the direction of the peritoneal cavity, in which case a pelvic abscess forms.

If the patient does not succumb to the infection or its results, a complete restitution, histologically and functionally, may occur. The infective bacteria are then killed, the foci are absorbed, the pus subsides and scar tissue formation completes the healing.

In a great number of cases, however, complete recovery does not occur. Resolution is slow and partial. A proliferation of the intermuscular connective tissue ensues. The acute metritis has passed to the chronic form.

The *symptoms* are *rigors, pyrexia,* and *pain,* together with those of endometritis, especially *pyorrhea.* The initial rigor and pyrexia are marked. The pain is felt in the pelvis and sacrum and is of a colicky or lancinating character. There are painful urinations and defecations. The discharge is foul-smelling and purulent. Involvement of the peri- and parametrii calls forth additional symptoms. Septicemia and bacteriemia may supersede, masking the local condition.

The *signs* are *enlargement* and *sensitiveness* of the uterus and *profuse pyorrheal discharge* from the cervix, which contains the causative bacterium.

The *prognosis* is invariably grave, depending upon the kind, virulence and number of the infective agents and the resisting power of the host. A persistent spreading of the infection increases the gravity of the prognosis, as do also the ever present danger periods in a woman's life which invariably cause exacerbations.

Treatment is 1. prophylactic, prevent infections; and 2. curative, consisting of rest in bed, ice coil to the abdomen, incision of abscesses if they form and can be located. Otherwise the treatment is symptomatic.

Chronic Metritis.

In the majority of cases chronic inflammation of the myometrium is the result of a neglected acute metritis, which was produced by a bacterial infection.

Teilhaber also recognizes a *primary simple chronic* or hyperplastic *metritis* characterized by an hypertrophy of the connective tissue. This is the fibrosis uteri of English writers. It does not depend on a bacterial infection. The non-inflammatory chronic metritis is caused by a hyperemia due to uterine displacements, chronic obstipation, ungratified sexual desire, interrupted coitus, impotency of the husband, sedentary life, want of physical exercise and cardiac, renal and hepatic disease.

Pathology. The uterus is more or less enlarged. Its consistency is hard and firm. Upon incision a marked grating and resistance may be noted. The cut surface is whitish on account of the preponderance of connective tissue. The vessels are sclerotic, protrude above the cut surface, and are increased in numbers.

Microscopically a pronounced increase in connective tissue is seen, which stains red with Giesson's method. The vessel walls have hyaline degenerations showing calcareous plaques in places. Lymphocytic infiltration and plasma cells are seen in the infectious form, but they are never present in the hyperplastic or simple catarrhal form.

Symptomatology. The onset is insidious. There is usually a history of a preceding infection. If the metritis follows a puerperium, the patient states that she has never felt well since her last labor.

The *characteristic symptoms* are *pain, profuse secretion* and *menorrhagia.* The pain is located in the sacral region. There is a dragging sensation in the deep parts of the pelvis. Vesical tenesmus and obstinate constipation are usually present. The uterine discharge is either

serous or purulent and causes burning and often pruritus of the vulva. The menorrhagia is profuse. The blood is arterial and often forms clots. The passing of the coagulæ may cause dysmenorrhea.

General disturbances of health, loss of enjoyment of life and earning capacity are the inevitable consequences. Secondary anemia almost invariably follows. There is loss of weight and strength, dyspepsia, anorexia, headaches and a feeling of weight and heaviness in the lower abdomen. Sterility may or may not be present. If conception takes place it is often interrupted.

Signs. Bimanual examination elicits an enlarged uterus of hard and firm consistency. The uterus is intensely sensitive. In the terminal stages the organ may be small, hard and insensitive. Very frequently a sensitiveness of the recto-uterine pouch, along the sacro-uterine ligaments, can be elicited (chronic posterior parametritis).

The *differential diagnosis* considers pregnancy and myomata uteri.

The *prognosis* is influenced by the length of time of the existence of the disease. If treated early complete restitution to the normal is possible. Complications, as endometritis, unfavorably influence the course of myometritis.

A liability to cancer of the uterus is always present. The occurrence of the climacteric, with subsequent senile atrophy, influences chronic myometritis favorably.

Treatment. Prophylaxis should begin with a rational stimulation of post-puerperal involution by means of careful asepsis, the administration of ergot, hydrastis, nourishing diet, etc., general body care by massaging, hot vaginal douches, and Sitz baths.

In non-puerperal cases a rational hygiene during puberty and menstruation, with attention to bowels and

bladder, the avoidance of sexual excesses and a correction of abuses of the genital organs, should be taught.

The *curative treatment* should be preceded by a microscopical examination of the endometrium in order to exclude tuberculosis and malignancy. The treatment is then begun with scarifications, followed by the use of a Bier's cup and a vaginal tampon. Should the bleeding become too profuse a solution of iron chlorid is applied and the patient directed to remain on her back for several hours.

Sitz baths in plain water, salt water, bran water or decoctions of oak-bark give marked relief. The temperature of the bath should be 90 degrees F.; the duration ten minutes. Some women bear cooler Sitz baths at 70 degrees F. better. The patient is put to bed after such a bath. Pain is relieved by cold applications or Priessnitz compresses to the lower abdomen.

Coitus must be prohibited. After six to eight scarifications have been made, iodin, tannin or ichthyol may be used locally. Tincture of iodin is applied to the endometrium, the fornix and portio, while a ten percent. solution of tannin or ichthyol in glycerin is placed against the cervix and fornix by means of a tampon. Vaginal douches of solutions of iodin (℞ Pot. iod. 5.0, tr. iodin 25.0, Aq. q. s. ad. 175.0. M. S.: Use one tablespoonful in three quarts of water), sodium chlorid (4.0 gm. to one litre of water) or sodium bicarbonate (4.0 gm. to one litre of water) should be made daily. It also is necessary to regulate the function of the bowels by a proper diet, the careful administration of cathartics and the use of gymnastic exercises. The menu should be reversed from its customary order (begin with fruits and vegetables). Such a course of treatment should last from six to eight weeks. The taking of baths at watering places (West Baden, Mount Clemens, Waukesha) or a change

of climate (mountain and woods) are very beneficial after the termination of the local treatment.

The menorrhagia should be treated by iodothyrin (0.2 gm. t. i. d.), the anemia by iron and arsenic (0.002 t. i. d.). In elderly women near the menopause intensive x-rays or radium should be used to bring about a cessation of menstruation. Actino-therapy renders hysterectomy unnecessary.

Myocervicitis.

Synonyms: Hypertrophy of the cervix; metritis cervicis.

We recognize several causative factors, viz., 1. hypertrophy of the supravaginal portion due to traction from a prolapsed vagina causing elongation of the cervix. 2. Hypertrophy or elongation of the vaginal portion due to a stenosis of the external os, resulting in an overdistention of the cervical canal by a retention of the discharge from an endocervicitis. 3. It may follow an acute endocervicitis. 4. Laceration of the cervix frequently terminates in a hypertrophy, the result of a chronic inflammation.

The *pathology* is the same as that of chronic myometritis corporis.

The *symptoms* are *pain* and *profuse leucorrhea.* The former is felt as a burning sensation in the sacral region and in the depth of the pelvis. Vesical tenesmus and obstinate constipation are usually present. The leucorrhea is white or yellowish, mucoid or purulent, and of an aqueous or creamy consistency.

General disturbances of health, sterility and interrupted pregnancies may be present, just as in chronic myometritis.

Signs: Inspection reveals an enlarged, edematous and hard cervix. A profuse secretion gushes forth from the cervical canal. Papillary and follicular erosions and

ectropion of the cervical mucosa may be seen. These result from the coexisting endocervicitis.

The *prognosis* is the same as that of chronic myometritis.

The *treatment* also is the same as that recommended for chronic myometritis, except that a high amputation of the cervix is indicated if medical treatment fails to bring about a cure.

Membranous Dysmenorrhea.

Dysmenorrhea membranacea, or exfoliativa means the expulsion of the entire, or large portions of the, menstrual decidua during menstruation. The expulsion is accompanied by intermittent, labor-like pains and profuse hemorrhages.

The *etiology* is unknown.

Pathology. The surface epithelium of the endometerium is found destroyed. The stroma cells are enlarged and stain poorly. There is a marked leucocytic and lymphocytic infiltration and a serous permeation of the entire mucosa. The glandular epithelium is usually exfoliated.

Symptoms. Severe colicky pains are felt at the time of menstruation. The latter is preceded by a feeling of fullness and general malaise. The expulsion of the decidua is accompanied by profuse bleeding. Menstruation is usually delayed for two weeks or longer. Sterility is present. If pregnancy takes place it is often prematurely terminated.

The *diagnosis* is made by a microscopical examination of the tissues expelled during menstruation.

Treatment. The disease is very obstinate. Repeated curettages, followed by cauterization of the endometrium with tincture of iodin, liquor ferri chloridi or ten to fifty percent. aqueous solutions of zinc chlorid, may be employed.

Tuberculosis of the Female Genital Organs.

Tuberculosis of the female genital organs is either a primary or secondary disease. The percentage of tuberculous infections of all the purulent diseases of the genital organs varies in different localities and may be from 7 to 24 (Menge, Martin). A positive diagnosis should not be made without confirmation by the microscope.

The routes of infection are either hematogenous, descending or ascending. The hematogenous and descending infections invariably are secondary, while the ascending infection is always primary. If we can demonstrate besides the genital tuberculosis another tuberculous focus in the body we must consider the genital the result thereof. Tuberculous infections of the female genitalia are usually secondary.

We distinguish two forms of genital tuberculosis: 1. The miliary, characterized by the formation of epithelial tubercles and giant cells and 2. the cheesy infiltrating form characterized by a caseous degeneration.

The tubercle bacillus can always be demonstrated in microscopical sections by the Ziehl-Neelsen method if the tissues are properly hardened in formalin alcohol. However, the bacillus is with difficulty found in the transudates and exudates of such diseased tissues unless a culture is made.

Of the different parts of the genital organs the tubes are most frequently invaded by tuberculosis, next the corpus uteri, more rarely the cervix and ovaries, and lastly the vagina and vulva.

Tubal Tuberculosis.

This may be primary or secondary and is either miliary or caseous.

Pathology. The *miliary form.* The mucosa is invaded first and shows the usual picture of a catarrhal inflam-

mation with typical epitheloid tubercles and giant cells. The presence of the tubercle bacilli can usually be demonstrated. The tube is moderately thickened and the muscular and serous coats are entirely free from tuberculosis in this beginning stage.

In advanced cases the mucous membrane is found destroyed, and the tube is filled with a purulent mass derived from the cheesy degeneration of the tubercles, while the muscular and serous coats are studded with tubercles. The tube is very much enlarged and tortuous but the fimbriated extremity usually remains open. The process may heal, leaving fibrous nodules behind, while the mucosa undergoes an atrophy.

The cheesy form. The entire tube forms a sac which sometimes attains the size of a child's head. It is filled with a cheesy substance. The muscular and mucous coats are entirely destroyed and the opening through the fimbriated extremity obliterated. The tube is adherent to the surrounding organs, in which tuberculous foci are deposited.

A rupture of the tubal sac may occur. The contents then discharge either into the peritoneal cavity, the intestines or the bladder. Other pathogenic bacteria, especially streptococci and colon bacilli, may thereafter invade the tubal cavity, setting up a mixed infection. The tubercle bacilli are found in great numbers in the cheesy tubercles.

Uterine Tuberculosis.

Uterine tuberculosis occurs much more frequently than commonly supposed. It usually is accompanied by tubal tuberculosis. However, primary tuberculosis of the uterus without participation of the tubes has been observed.

Both the miliary and cheesy forms are encountered. In the former we find an interstitial inflammation, hyper-

emia, round-cell infiltration, and tubercles with giant cells and tubercle bacilli.

In the cheesy form the endometrium is rapidly destroyed and the process penetrates deeply into the myometrium. The uterine cavity is then filled with a cheesy substance and the entire organ becomes much enlarged.

Uterine tuberculosis remains ordinarily confined to the body of the organ, yet it may invade the cervix or be limited to the latter. Cervical tuberculosis is usually of the caseous form. On the portio vaginalis tuberculous ulcers with a dirty coating may be seen, also cauliflower-like tumors occur, which rapidly undergo degeneration.

Vaginal and Vulvar Tuberculosis.

Tuberculosis of these organs is very rare, the squamous surface epithelium affording a natural protection. It is more often secondary than primary.

In the vagina tuberculosis occurs as a flat ulcer which has indurated edges and tends to extend slowly.

In the vulva large-sized, deeply-seated ulcers occur which frequently form fistulæ. They may extend upward into the vagina. The edges of the ulcers are undermined, the connective tissue stroma surrounding the ulcer increases and the labia become elephantoid.

Ovarian Tuberculosis.

Tuberculosis of the ovary is very rare and as a rule secondary. The miliary form is the one usually present, the tubercles being confined to the cortex. Ovarian cystadenomata also may become infected with tubercle bacilli.

Peritoneal Tuberculosis.

Peritoneal tuberculosis is very frequent and usually occurs in the young. It is always secondary. The infection is carried to the peritoneum either by lymph vessels

and glands from intestinal tuberculous foci, or through the stomata of the diaphragm from pulmonary and pleural foci or, though very rarely, from the genital organs.

Two forms are distinguished: (1) the diffuse miliary form which is characterized by marked ascites, and (2) the dry form in which cavities are found that were formed through adhesions with the surrounding organs. These cavities become filled with a serous, caseous material. The abscesses thus formed rupture into the contiguous viscera.

The *symptoms* of tuberculous disease of the genitalia are the same as those observed in the septic infections plus those characteristic of a tuberculous infection.

Diagnosis. The secondary form is easily diagnosed if the primary focus can be detected. The diagnosis should be verified by the miscroscope and eventually a histological test in the guinea pig. The general constitutional symptoms, such as the afternoon temperature, the absence of a leucocytosis (while a leucocytosis in tuberculous peritonitis is the rule) should be taken into consideration. The tuberculin diagnostic test may give positive data. 0.003 cc. of Koch's old tuberculin should be used at the first injection, and 0.005 cc. for the second. If a reaction, either general, (malaise, rise in temperature) or local at the point of injection, (redness, tumefaction, pain) or focal at the seat of lesion, (increase of pain, increase in size of infection) occurs, the diagnosis is affirmative.

Treatment. Secondary genital tuberculosis should be treated conservatively by the usual anti-tuberculous methods. Surgical treatment of secondary genital tuberculosis leads almost always to an aggravation of the primary disease.

If the tuberculosis is manifestly confined to the genitals, and if it causes the bearer to become an invalid, and

further, if the usual constitutional anti-tuberculous treatment cannot be carried out on account of sociological conditions, we may have to decide on surgical treatment. This must be as radical as possible and consists of a panhysterectomy. The result of surgical treatment of mixed infections is usually much more unfavorable than that of purely tuberculous infections.

Tuberculosis of the vulva calls for a total excision of the part. Miliary tuberculous peritonitis necessitates a simple evacuation of the ascitic fluid through a laparotomy incision. In the dry form of tuberculous peritonitis operations are contra-indicated. In all inoperable cases applications of intensive x-rays are very beneficial.

CHAPTER VII.

TUMORS OF THE FEMALE GENITALIA.

General considerations. New growths of the female genital organs occur either in the vulva, vagina, uterus, tubes, ovaries, or para- and perimetrium.

Tumors of the vulva, vagina and uterus will be taken up in this chapter. Neoplasms of the tubes, the para- and perimetrium, are so rare that they will not be discussed. Primary cancer and embryonal tumors are the most common new growths found in the tube. Tumors of the ovaries will be found described in another chapter.

Tumors form by proliferation of (a) epithelial cells, (b) connective or desmoidal tissue and (c) embryonal or ovigenous tissue. They are either benign or malignant.

Etiology. We do not know anything definite about the direct cause of the tumors, except the embryonal variety.

Tumors of the Vulva.

The benign *epithelial cell* tumors of the vulva are the cystomata (Bartholin's gland) and the urethral caruncle; the malignant type are the carcinomata. The benign *connective tissue* tumors are the fibromata and lipomata; the malignant, the sarcomata. *Ovigenous* tumors of the vulva have been met with but they are so rare as not to possess practical clinical importance.

The *general characteristics* of vulvar new growths are, (1) they are very infrequent, (2) they are more often

malignant than benign, and (3) they cause discomfort in walking, urinating and cohabiting even if they are of a small size.

I. Ephithelial-Cell Tumors of the Vulva.

1. Cystoma Vulvæ.

Cysts originate in Bartholin's gland and are usually unilateral, though they may be bilateral. They are retention cysts resulting from an inflammatory obstruction or obliteration of the gland duct. The walls of the cyst are composed of a smooth, stratified, cylindrical epithelium, the contents being a mucus glandular secretion. Such cysts may grow to a considerable size.

2. Urethral Caruncle.

This tumor is found at the meatus urinarius where it forms a small, fleshy excrescence the result of a simple irritative hypertrophy of the mucosa. It is very vascular, bleeds easily, is very tender and has a bright-red color. A caruncle may become covered with urinary or bacterial deposits of a whitish-gray color.

3. Carcinoma Vulvæ.

Carcinomata of the vulva originate from the clitoris and urethral orifice. Those growing from the clitoris form squamous, epithelial-cell cancers, those from Bartholin's gland cylindrical epithelial-cell cancers. Secondary infections of such cancers are the rule, the infectious material being derived from the vagina. Such tumors grow rapidly, disintegrate early and extend into the neighboring organs (vagina, bladder, rectum) forming urinary or fecal fistulæ. Chronic inflammations and kraurosis of the vulva predispose to cancers.

II. Connective Tissue Tumors of the Vulva.

1. Fibroma Vulvæ.

Fibromata are of rare occurrence. They may originate in all parts of the vulva and especially the labia majora, where they are derived from the round ligament. Such neoplasms may grow to an enormous size. They frequently become pedunculated. The peduncle may stretch and thus obstruct the blood supply of the tumor. The latter then undergoes a necrosis and becomes detached from the body. Fibromata may undergo a sarcomatous degeneration. They should be differentiated from elephantiasis vulvæ.

2. Lipoma Vulvæ.

Lipomata also are rare and grow in the labia majora and mons veneris. They frequently are of a large size and may become pedunculated. Torsion or traction of the peduncle may lead to necrosis.

3. Sarcoma Vulvæ.

Sarcomata of the vulva are very rare and are usually of the melano-sarcomatous type. They disintegrate early, invade neighboring tissues and organs and behave very much like the carcinomata.

The *symptoms of the benign tumors* of the vulva are of a mechanical nature and consist in an interference with walking, urination and cohabitation. The urethral caruncle causes dysuria and even ischuria.

The *diagnosis* of tumors of the vulva is made by inspection, palpation and microscopical examination.

The *differential diagnosis* concerns tumors, hernia of the labia (especially of the round ligament), hydrocele and inflammatory neoplasms. See Table No. 1, page 98.

Treatment of the *benign tumors* is purely surgical.

The pedicle of pedunculated tumors should be ligated

and severed. The removal of all other tumors is performed as follows: An incision is made on the skin surface. The tumor is peeled out by a blunt dissection, either with the Mayo scissors or by a dry gauze dissection. All bleeding points should be carefully looked for and ligated. The bed of the tumor should be obliterated by transverse, interrupted, purse-string, catgut sutures. Finally the

Table No. 1—Differential Diagnosis of Benign Tumors of Vulva.

	Benign tumors of the vulva.	Inguinal hernia.	Hydrocele.	Inflammatory tumefaction.
Onset.	Insidious.	Sudden, after trauma.	Sudden, without apparent cause or trauma.	Sudden, after an infection.
Impulse on coughing.	No.	Yes.	No.	No.
Reducible.	No.	Yes.	No.	No.
Pain.	No, unless from pressure.	No, unless irreducible.	No.	Yes.
Percussion.	Flat; fluctuation if cystic.	Resonant.	Fluctuation.	May have fluctuation with abscess formation.
Discoloration.	No.	No.	No.	Yes. Reddish.
Temperature.	No.	No.	No.	Yes.
Leucocytosis.	No.	No.	No.	Yes.
Course and termination.	Grows slowly.	Variable, may grow larger.	Variable, may increase in size.	Disappears after suppuration or organization.

skin incision is closed and the wound dressed with Beck's paste dressing.

A urethral caruncle is snipped off with scissors. The urethral and vulvar mucous membranes are then coaptated by sutures, preferable catgut. A retention catheter should be inserted for from five to six days.

The *symptoms of the malignant tumors* are interference with walking, severe pain during urination or cohabitation, profuse discharge, hemorrhage when the

growth breaks down, and symptoms of infection. Invasion of the regional lymph glands and neighboring organs cause additional symptoms. The terminal stage is characterized by cachexia.

The *diagnosis* is made from the history of the case, inspection, palpation and microscopical examination. Abderhalden's serum diagnosis may be of value.

The *differential diagnosis* from luetic and tuberculous ulcers and tumors is made from the history of the case, the clinical entity, the microscope, and Wassermann, luetin and tuberculin tests.

The *treatment* differs depending on the operability or inoperability of the condition.

Operable cases indicate a radical extirpation of the entire vulva, with the removal of the inguinal and femoral lymph glands of both sides and of the subcutaneous adipose tissue in one mass. The involved parts of the vagina and urethra should also be removed. This excision should be followed by actino-therapy (radium and massive x-rays) to prevent recurrence.

Inoperable cancers should be treated with radium and massive x-rays.

Hopeless cases may be treated symptomatically. Hemorrhage may be arrested with the cautery iron at a dull heat. Pain is relieved by opiates and the general strength kept up by the administration of tonics (Bashham's mixture 4.0 cc. t. i. d.), nux vomica (0.3 cc. of tincture t. i. d.) and arsenic hypodermically (salvarsan or sodium caccodylate).

Tumors of the Vagina.

The benign *epithelial-cell* tumor of the vagina occurs as a cystoma, the malignant as a carcinoma; and the benign *connective tissue* neoplasm as a fibroma, the malignant, as a sarcoma. *Fetal cell inclusions* in the vagina are exceedingly rare.

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Vaginal new-growths are of rare occurrence. They resemble those of the vulva on account of the same general histological structure.

Ephithelial Tumors of the Vagina.

1. Cystoma Vaginæ.

Two varieties are recognized: (a) retention cysts and (b) cysts of Gärtner's ducts.

The *retention cysts* form either by an agglutination of adjoining folds or by the occlusion of an aberrant mucous gland of the mucosa. Such cysts may attain the size of a hen's egg. The lining is composed of either stratified squamous or cylindrical cell epithelium (from mucous glands). The contents are a clear, tenacious fluid, rich in albumin.

Gärtner's cysts are located in the anterior or anterolateral portion of the vaginal wall. They are lined with stratified cylindrical cell epithelium and contain a clear, serous fluid like the par-ovarian cysts.

2. Carcinoma Vaginæ.

Primary malignant epithelial neoplasms of the vagina form squamous cell cancers. They are either circumscribed ulcers or diffuse infiltrating growths. Disintegration occurs early. Invasion of neighboring organs is rapid and the formation of metastases in the deep inguinal and iliac lymph nodes occurs early.

Connective Tissue Tumors of the Vagina.

1. Fibroma Vaginæ.

Fibromata originate in the walls of the vagina and may locate anywhere in the canal. They cause a bulging of the vaginal wall and may form decubital ulcers, the result of pressure. The consistency of these desmoids

varies from hard to soft. Softening is caused by a lymph stasis. These tumors never fluctuate (cysts do).

2. Sarcoma Vaginæ.

Primary vaginal sarcomata are the rarest of the tumors of the female genital canal. They occur at all ages, in childhood as well as adult life, and old age. The sarcomata may be round- or spindle-celled. They originate in the vaginal wall, proliferate rapidly into neighboring structures and have a tendency to an early superficial ulceration. They form either an ulcer or a solid growth.

The *symptoms of the benign vaginal tumors* are purely mechanical. Small tumors are symptomless. Larger ones cause pressure-pain, interference with cohabitation and the sensation of a descent or prolapse of the walls.

The diagnosis is made by inspection and palpation. The neoplasms must be *differentiated* from cystocele and rectocele. If a catheter is inserted into the bladder and a finger placed in the vagina a tumor may be recognized from a cystocele. If a finger is inserted into the rectum and another into the vagina, a tumor of the posterior vaginal wall may be differentiated from a rectocele.

Treatment consists in an excision of the neoplasm. The incision should be made through the mucosa over the tumor, which is then peeled out by blunt dissection. Guard against a perforation into the bladder or rectum. All bleeding points should be ligated. The bed of the tumor is closed by suturing with catgut. The wound in the mucosa should be coaptated by stitches. Finally a tampon of iodoform gauze saturated with Beck's bismuth paste may be inserted into the vagina and removed on the fifth day following the operation.

The symptoms of malignant tumors consist of those of the benign growth plus profuse, putrid discharge of a reddish, watery color, hemorrhage and pain.

The diagnosis is made by inspection, palpation (which

elicits the irregular, ill-defined and infiltrating character of the growth) and the microscope.

The differential diagnosis concerns tuberculosis and luetic ulcers or gummata and is made by the microscope, the Wassermann and luetin reactions, and the Abderhalden serum diagnosis.

The *prognosis* is grave.

The treatment varies, depending on the operability or inoperability of the growth.

An operable cancer may be eradicated by a radical removal of the vagina, uterus, adnexa, parametrium and pelvic lymph nodes. The operation should be followed by the prophylactic use of massive Röntgen rays and radium to guard against recurrences.

An inoperable vaginal cancer may be treated with radium and x-rays.

Hopeless cases should be treated symptomatically by vaginal douches of zinc chlorid solutions (℞ Zinci chloridi 30.0, alcoholis 75% ad 175.0, M. Signa: Use one teaspoonful in two quarts of water as a douche), packing the vagina with calcium carbid or acetone (Gellhorn), to remove the odor and stop the discharge. Pain may be relieved by x-rays or opiates and other symptoms by the indicated remedies.

Tumors of the Uterus.

Uterine newgrowths are the most frequent of any tumors in the female body.

The *epithelial-cell tumors* of the uterus are, as a rule, malignant. They are the carcinomata and chorion epitheliomata. The *connective tissue tumors* of the uterus are benign and malignant, the benign tumors being the myomata and the malignant the sarcomata. *Embryonal tissue tumors* of the uterus are, indeed, of rare occurrence.

Let us discuss the uterine neoplasms not according to

the histological classification, but according to their clinical importance and frequency of occurrence.

1. Myomata Uteri.

Synonyms: Fibroids, fibromata, fibromyomata.

Pathology. Myomata uteri are composed of the elements of the uterine wall. These are *connective tissue* and *muscle fibres.* The one or the other element may predominate. The tumor is hard, white and poor in blood-vessels if connective tissue predominates, and soft, juicy, reddish and vascular if muscle surpasses the amount of connective tissue. The tumor is always *encapsulated,* the capsule being formed by the compression of the elements of the uterine wall surrounding the tumor. This capsule distinctly separates the growth from the myometrium and also facilitates its enucleation. A solitary myoma is the exception, usually a great number of tumors being found. The size of a single myoma may vary from a pinhead to a mass weighing forty pounds. The myomata are mostly located in the corpus and very rarely in the cervix.

According to location we distinguish four varieties of *corporeal myomata.* 1. Subserous, 2. Submucous, 3. Intramural or interstitial and 4. Intra-ligamentous.

Subserous and submucous myomata may become pedunculated. Torsion of the pedicle causes stasis, leading to necrosis and finally a complete separation of the tumor from its host. A pedunculated submucous myoma acts like a foreign body in the uterine cavity, and excites the uterus to contraction, thus becoming expelled into the vagina.

Intramural tumors may grow towards the serous or mucous layers, changing into the subserous or submucous form, when they assume the characteristics of these varieties.

Intra-ligamentous myomata develop from the lateral

wall of the uterus into the folds of the broad ligament. They usually cause pressure symptoms on the bladder, rectum or ureter on account of their immovability. They may also become detached from the uterus.

We distinguish three varieties of *cervical myomata:* 1. Supra-vaginal, 2. Intra-vaginal and 3. Pedunculated submucous myomata. The supra-vaginal myomata are rare and either displace the bladder or rectum or grow upwards and laterally into the broad ligament. The intra-vaginal is the rarest location for a cervical myoma, while the pedunculated submucous myomata are the most common of cervical myomata. The latter originate in the cervical canal, are expelled into the vagina and usually become pedunculated, when torsion, necrosis and detachment may occur.

The *general characteristics* of myomata uteri. Myomata occur during the child-bearing period of a woman's life, mostly between the thirtieth and fiftieth years. Their growth is slow and gradual unless complications, especially degenerations, cause a rapid increase in size.

Spontaneous involution during the puerperium and climacteric is possible; however, the opposite may, likewise, take place.

Adenomyomata uteri contain connective tissue, muscle fibres and glandular tissue. The glandular tissue forms an inclusion, being derived from either the uterine mucosa (mucous adenomyomata) or from the epoophoral cells derived from the parovarian tubules (epoophoral adenomyomata). The latter are located in the corpus or cornua uteri.

Characteristics: Adenomyomata do not possess a capsule. They grow diffusely into the uterine tissue and usually remain small. Their diagnosis can only be made microscopically.

The influence of myomata on the uterus. The form of the uterus changes, corresponding to the size, number and

location of the neoplasms. The cavity becomes deformed or tortuous with intramural and submucous myomata. The uterine wall hypertrophies and the mucous membrane increases its surface extent and thickens.

The *influence of myomata on the endometrium.* The continued irritation produces an hypertrophy of the glands and the interstitial tissue and a hyperemia. The pressure of the tumor may cause an atrophy of the mucosa over the vertex of the growth.

Degenerations of myomata. Four per cent. of the myomata become *malignant, i. e., sarcomatous.* Carcinomatous degeneration may occur in an adenomyoma. Malignant degeneration is accompanied by rapid growth of the neoplasm, the formation of extensive metastases and an early cachexia. *Necrosis, gangrene and infection* may result from stasis due to torsion. Infection chiefly occurs in submucous and intramural growths. *Telangiectasis or lymphangiectasis* is caused by circulatory disturbances resulting in hemorrhage within the tumor, an extensive destruction of tissue and a rapid increase in the size of the growth. *Atrophy* and *calcification* have been observed. They must be considered as a spontaneous cure. *Fatty degeneration* is frequent, and occurs especially after pregnancy and during the puerperium. The tumor assumes an homogenous, yellowish color. *Myxomatosis* leads to a rapid increase in the size of the tumor and a gradual liquefaction. The fluid is hemorrhagic, the capsule remaining so that the mass resembles a cyst. *Necrobiosis* occurs if the circulation of blood in the tumor becomes obstructed and micro-organisms do not gain access to it. Such a tumor is soft, edematous and brownish-red.

Symptoms. General remarks. Colored and mulatto women suffer more from myomata than Caucasians. Women of the better walks of life more frequently suffer from myomata than the poor, hard-working women with

many children. Vice versa, the latter show a greater percentage of cancers than the former. Myomata occur oftener in unmarried and nulliparous women than in multiparæ. The intensity of the symptoms does not correspond to the size and number of myomata, but apparently depends on the changes that take place, (1) in the endometrium (hyperplasia and hypertrophy), (2) in the ovaries (hyperplasia, retention cysts and follicular hematoma) and (3) in the heart (fatty and brownish myocarditis).

The cardinal *symptoms* are (1) hemorrhage, (2) pain, (3) profuse discharge and (4) usually sterility.

Hemorrhage is due to a gradual increase from month to month, or year to year, in the duration, quantity and frequency of the menses, and a change in the character of the flow (clots). The climacteric is postponed, often to the end of the fifth decade. The results are chronic anemia, a feeble and rapid pulse, lowered blood pressure and a decrease in the percentage of hemoglobin to thirty or less. The *pain* is either a dysmenorrhea or a pressure-pain. The pressure-pain is caused by the weight or impaction of the tumor, leading to a compression of the urinary and intestinal canals, a pressure on the nerves and an edema of the lower extremities resulting from an obstruction to the blood circulation. The *leucorrhea* is brought about by the endometritis. The *sterility* is due to the changes in the endometrium and ovaries.

Degeneration of the tumor and torsion of the pedicle lead to a rapid increase in size of the tumor, an aggravation of the intensity of the pain and an alarming increase in the uterine hemorrhage. Infection and suppuration are characterized by an initial rigor, pyrexia and pyemic symptoms.

The influence a myoma might assert on a pregnant uterus depends on the location and size of the tumor.

	Hematocele.	Perimetritis.	Displaced abdominal organs.
History.	idden onset. rauma, operation, tubal pregancy.	Sudden onset after infection following labor or operation.	Pertains to function of displaced abdominal organ (spleen, kidney).
Symptoms.	ock. Severe nemia. Peritoeal irritation.	Acute form, fever and peritoneal signs. Later, pains due to adhesions.	Depend on organ involved.
Signs. Uterine position.	ıte-posed.	Ante-posed.	Uterus and adnexa negative. Palpate abdominal organ, which is missing in normal place. May be replaced into normal position.
Size and form.	rmal.	Normal.	
Movability.	xed.	Fixed.	
Consistency.	rmal.	Normal.	
Sensitiveness.	gative.	Sensitive.	
Adnexa: position.	ass posterior in ul-de-sac, inluding adnexa nd displacing ectum.	Mass post. to uterus including adnexa and rectum. Bulging of post. vault. Decreases gradually or leads to suppuration.	
Form.	ılging into post. ag. vault.		
Sensitiveness.	ecreases gradally with orranization.		
Size.			
Consistency.			
Growth.			
Laboratory exam. Microscopic.	egative.	Negative.	
Blood.	nemia.	Negative, except leucocytosis in acute form.	Negative.
Abderhalden's serum diagnosis.	egative, except n tubal pregancy.	Negative.	Negative.

	atocele, Perimetritis.	Nephroptosis, Splenoptosis.
History.	s hematoma.	Insidious onset.
Symptoms.	s hematoma.	Interference in urination as Dietl's crisis. Feel wandering of organ.
Signs. Uterus.	imbedded in mass and vable. Bulging of post. al fornix.	Uterus negative.
Adnexa.	adnexa imbedded in	Negative.
Sacrouterine.	apart.	Negative.
Rectum.	ssed by exudate.	Negative.
Blood.	s hematoma.	Negative.
Abderhalden.	s hematoma.	Negative.
X-ray.	ve.	Negative.

The latter may either hinder the development of the ovum or prevent its expulsion during labor.

The influence of pregnancy on the tumor consists in a fatty degeneration and softening, while involution of the genital organs during the puerperium may cause an atrophy of the myoma.

The diagnosis is made by palpation. If the tumor is palpable the diagnosis is easy, especially if the symptoms are considered, particularly the menorrhagia. A diagnostic curettage and microscopical examination must be performed in each case to determine malignancy.

Differential diagnosis of a *small myoma,* the size of a man's fist, comprises (1) pregnancy, (2) cancer of the corpus uteri, (3) tubal disease, (4) ovarian tumors, (5) parametritis, (6) perimetritis, (7) hematoma, (8) hematocele and (9) displaced abdominal organs (kidney, spleen). *Medium-sized uterine tumors* should be differentiated from (1) pregnancy of the second trimester, (2) medium-sized ovarian tumors, (3) parametritic and perimetritic exudates, (4) pelvic hematoma or hematocele and (5) displaced abdominal organs. *Large myomata* should be differentiated from (1) large ovarian cysts, (2) ascites, (3) tympanites, (4) phantom tumors and (5) pregnancy of the third trimester. (See tables 2, 3 and 4.)

The *prognosis* of uncomplicated myomata, if they are correctly treated, is favorable. Anemia, myoma heart, and the character of the degeneration make it less favorable or even unfavorable.

Treatment. (1) Myomata not causing symptoms do not require any treatment. (2) If they cause symptoms, but are uncomplicated and not of the submucous or cervical varieties, and the patient is near the fortieth year of life, then massive x-raying or, better still, radium treatment after a diagnostic curettage, is the course of choice. (3) Submucous and cervical myomata causing

Table No. 4—Differential Diagnosis of Large Tumors.

	Colossal ovarian tumor.	III. Trimester pregnancy.	Colossal myomata.	Ascites.	Other abdominal tumors.
History.	None. Insidious onset. Very slow enlargement.	Symptoms of pregnancy, especially amenorrhea.	Gradually increasing menorrhagia.	Hepatic, nephritic or cardiac disease.	Referable to organ diseased.
Symptoms.	Pressure and weight.	Amenorrhea, nausea, enlarged breasts, quickening.	Dysmenorrhea, menorrhagia, leucorrhea, pressure pain.	Referable to primary disease.	Referable to underlying disease.
Signs. Inspection.	Dome-like contour of abdomen, not changing with change in position.	Same. Fetal movements. Uterine contractions. Lightening.	Nodular, irregular mass. No change with change of position.	Oval outline. Changes with change of position.	Corresponds to organ enlarged. Mass moves with diaphragm if from upper abdomen.
Palpation.	Uniform consistency.	Same. Palpate fetus and fetal movements.	Hard, irregular.	Negative.	Negative except we outline organ.
Percussion.	Dullness over tumor. Tympanites laterally and above.	Same.	Same.	Tympanites centrally. Dullness laterally. Changes with position. Fluctuation wave.	Dullness continues towards origin of tumor.
Fluctuation.	Fluctuation wave.	Absent.	Absent.		Absent.
Auscultation.	Negative. Perhaps bruit.	Fetal heart-beat and movement; bruit.	Negative. Perhaps bruit.	Negative.	Negative. Perhaps bruit.
Bimanual.	Uterus palpable post. or ant. to tumor. Normal size and consistency.	Cervix soft and continuous with tumor mass.	Tumor contained within uterine wall. Cervix hard.	Genital organs normal.	Genital organs normal.
X-ray.	Negative.	Reveals fetus.	Negative.	Negative.	Negative.
Abderhalden.	Negative to placental tissue.	Positive.	Positive to myoma tissue, negative to placental tissue.	Negative.	Negative.
Hemanalysis.	Negative.	Negative.	Secondary anemia result of continuous menorrhagia.	Negative.	Depends on underlying disease.

symptoms should be removed by operation (myomectomy or hysterectomy). Intramural, intra-ligamentous and subserous myomata causing symptoms, but occurring in young females before the thirty-fifth year, should be removed surgically unless contra-indications to an operation exist, in which event the treatment should be the same as given under (2). If surgery is indicated the tumor should be enucleated or the uterus removed supravaginally. The ovaries should be left intact. Myomectomy tends to preserve the functions of menstruation and often also of conception and child-bearing. (5) Myomata undergoing degenerations should be subjected to surgical removal. (6) Hemorrhage, causing life-threatening symptoms, should be treated like any other uterine flow.

Carcinoma Uteri.

General remarks. The uterus is more frequently afflicted with cancer than any other organ of the female body. Cancer of the uterus stands next in frequency to that of myomata uteri.

The varieties of uterine carcinoma according to location and origin are as follows:

1. *Carcinoma of the cervix.* A. Of the portio vaginalis (a) from the squamous epithelium and (b) from the cylindrical epithelium of erosions. B. From the cylindrical epithelium of the cervical mucosa.

2. *Carcinoma of the corpus* (a) from the cylindrical surface epithelium of the corporeal mucosa, (b) from the cylindrical epithelium of the endometrial glands and (c) from submucous polypi of the endometrium.

Pathology. Carcinoma of the portio vaginalis derived from the squamous epithelium of the vaginal mucosa, forms numerous squamous-cell columns which penetrate into the connective tissue of the cervix. The columns of the epithelioma contain cancroids or epithelial pearls,

which possess areas of keratinization or cornification in the center.

If the carcinoma develops from the *cylindrical cells of erosions,* a cylindrical-cell cancer is found. The cylindrical epithelial cells may undergo metaplasia into squamous epithelium, when they adopt the same characteristics as the epitheliomata. The type of the cylindrical-cell carcinomata is glandular. The portio carcinomata form either (1) an *ulcer* surrounded by a wall-like border which is hard and densely infiltrated, (2) a *proliferating growth* which penetrates into the deeper structures, forming nodules therein and leaving the surfact intact, and leading to an early necrosis, or (3) a *cauliflower tumor* of great proliferating power, which grows rapidly into the vagina and attains a large size. The surface of the latter is uneven and nodular. Disintegration occurs early.

Carcinoma of the cervix originates from the glands of the cervical mucosa, and has the same characteristics as cancer originating from cervical erosions. It is a cylindrical-cell adenocarcinoma. Cervical cancers occur in two different forms either (1) as polypous tumors, or (2) as infiltrating growths. Both forms lead to a rapid destruction of tissue with the formation of a deep crater lined with disintegrating necrotic cancer masses.

Carcinoma of the corpus develops either from the surface epithelium, mucous glands or mucous polypi of the endometrium. Carcinoma of the surface epithelium may undergo a metaplasia into a squamous-cell cancer, when it adopts the same characteristics as an epithelioma. Corporeal cancer, however, is more frequently derived from the mucous glands of the endometrium. The glands undergo an increase in size and number, the cylindrical cells proliferate through the basement membrane into the surrounding tissue and involve the entire uterine wall, invading the peritoneal cavity.

Mucous polypi often undergo a carcinomatous degeneration. The malignant neoplasm at first remains confined to the polypus, but later grows unlimitedly through the entire uterine wall, and even into the peritoneal cavity.

Corporeal carcinomata are usually *adenomatous*. They are circumscribed in the beginning and diffuse later on. The surface disintegration or breaking down does not occur as early in these carcinomas as in cervical cancers. The connective and muscular tissues gradually disappear. The cancer is surrounded by an area of *round-cell infiltration*, which is the reaction of the normal tissues against the invader.

The superficial disintegration offers an opening for the entrance of pathogenic bacteria. Such *infections* are of great importance to the physician on account of the danger of carrying the bacteria to others, especially parturient women. Operations for carcinoma are hardly ever aseptic and the result of surgical operations is doubtful. The fever so frequently present in cancer patients is caused by the infections. Disintegration further may lead to erosions of blood vessels and hemorrhage results.

The different varieties of all these cancers may be determined in the early stages by a microscopical examination.

Cancer in its terminal stages invades neighboring organs, as the vagina, bladder, rectum, adnexa and peritoneal cavity, leading to corresponding additional symptoms. The pelvis finally becomes one mass of cancer.

Metastases form early. Portio cancers invade the hypogastric and internal inguinal glands; cervical cancers the hypogastric, internal inguinal, lateral sacral and external iliac glands; and corporeal cancers the external iliac, the inferior and superior lumbar glands. Metastases also form in other glandular organs of the body, as the liver, the breasts, distant lymph glands and so forth.

The practical importance of this knowledge lies in the imperative necessity of removing these glands during operations for the eradication of the cancer, though the gland-disease might be secondary to the infections mentioned. However, it is impossible to establish this fact macroscopically.

Malignancy. Cervical cancers are the most malignant, portio cancers somewhat less, and corporeal cancers the least. Metastases in the latter also occur relatively late.

Symptomatology. General remarks. Early stages of carcinoma are seldom observed. So-called pre-cancerous conditions predisposing to malignant disease are erosion of the cervix, chronic endocervicitis, laceration, chronic polypoid endometritis and so forth. Routine microscopical examinations of such pathological conditions will promote earlier correct diagnoses.

The general symptoms of cervical cancer are: The beginning is symptomless. The majority of these cancers occur in multiparæ and only about five per cent. in nulliparæ. The *cardinal* symptoms are *hemorrhage, discharge, pain* and *general constitutional derangement.* The *hemorrhage* is at first menorrhagic, but soon becomes metrorrhagic, appearing on the slightest irritation, such as physical exertion, gynecologic examination, cohabitation, defecation. The discharge is at first profuse and aqueous, gradually assuming the characteristic putrid odor and brownish color due to a disintegration of tissue and an admixture of blood. The *pain* varies according to the time of its occurrence, character and the invasion of adjoining organs. In some cases it appears in the beginning, in others in the advanced stages. The intensity may be described as a soreness and dull ache, or as an intermittent, gnawing-like, unbearable and lancinating pain. An invasion of adjoining organs, as the bladder, rectum and nerves, is accompanied by dysuria, rectal tenesmus and pains along the affected nerves.

The constitutional symptoms consist of insomnia, which usually appears early, total loss of appetite, increased sexual desire and finally cachexia (loss of weight and strength, sallow, ashy discoloration of the skin).

The diagnosis should be made with the microscope, assisted by inspection and palpation. Involvement of neighboring organs may be determined by cystoscopy and rectoscopy.

Corpus cancer. General symptoms. Carcinoma of the body of the uterus occurs in about ten per cent. of all uterine cancers. It usually appears after the menopause. A history of profuse leucorrhea and pruritus vulvæ, which has existed for years, is often given. Pre-cancerous conditions are chronic endometritides, especially of the polypoid variety. Nulliparæ are more frequently affected with body cancer than multiparæ.

The cardinal symptoms are *hemorrhage, discharge* and *pain.* Hemorrhage takes the form of the reappearance of the menses and rarely occurs after local irritations. The discharge is profuse, putrid and sanious, often of an intermittent character. The discharge collects in the uterine cavity and its expulsion is preceded by labor-like pains. The pain is an ache or of an intensely lancinating character and located in the abdomen, lumbar region and legs.

The *constitutional symptoms* are the same as those of cervix cancer (insomia, anorexia, increased sexual desire and finally cachexia).

The *diagnosis* is based on the microscopical findings, the globular enlargement of the uterus and the symptoms, especially the reappearance of the menses and the intermittent pain, accompanied by a gush of the contents from the cavity of the uterus (pyometra).

The *prognosis* of all the uterine cancers is absolutely grave, and it can only be improved by (1) educating the physician to make routine microscopical examinations of

tissues in all uterine hemorrhages or chronic inflammations, (2) inducing him to immediately refer suspicious cases to a specialist, (3) educating the public to consult the family physician on the occurrence of uterine hemorrhages. Uterine carcinomata will then reach the surgeon while they can yet be totally eradicated. An anatomic cure can only be expected from the removal of all cancerous foci and tissues, which is possible only in the early stages when the cancer is yet a localized disease.

The treatment is divided into that of (1) operable cancers, (2) inoperable cancers and (3) recurrent cancers. Each case of cancer should be subjected to an exploratory laparotomy if the slightest possibility of total eradication seems to exist, depending on the results of a careful examination. If on exploration of the abdominal cavity the lumbar lymph glands are found involved, or extensive invasions of adjoining organs and tissues, such as the bladder and rectum, have taken place, the case is hopeless. The operation of choice is the radical extended abdominal pan-hysterectomy (Wertheim), preceded by a cauterization of the uterus through the vagina with a red-hot cautery. During convalescence radium and deep x-ray treatments should be employed to kill all cancer cells which may inadvertently have been left.

Inoperable cancers should be treated exclusively by actino-therapy, i. e., a combined use of radium and the Röntgen rays. If these cannot be had then an excochleation of all cancerous tissue with the curette and a cauterization of the bed of the wound with the red-hot iron should be performed. A fifty per cent. solution of zinc chlorid should be applied to the crater, which then is packed with powdered sodium bicarbonate. A tampon saturated with Beck's paste (20 parts of bismuth subnitrate in 80 parts of vaseline) should be placed in the crater and in the vagina and removed after three days.

A retention catheter is inserted into the bladder and left for five days.

Otherwise the patient is to be treated *symptomatically*. Hypnotics may be given to relieve the pain. Douches of zinc chlorid (one teaspoonful of a 1.5 per cent. alcoholic solution in two quarts of water), or applications of acetone, or the insertion of calcium carbid or sugar into the crater, are beneficial to ameliorate the odor and lessen the amount of the discharge. General supportive treatment is indicated to keep up the strength of the patient. Chemo-therapy may be tried. Hypodermic injections of borcholin or radium solutions have been highly successful in a few isolated cases.

Recurrent cancers should be subjected to the same principles of treatment as operable and inoperable cancers. If the recurrence can be completely eradicated by an operation then that should be the procedure, otherwise it should be treated with radium, x-ray and chemo-therapy.

Malignant Chorion Epithelioma.

Synonyms. Deciduoma malignum, syncytioma malignum.

Definition. A malignant chorion epithelioma is a tumor originating from the epithelium of the chorionic villi, the remnants of a preceding pregnancy, especially a hydatid mole.

Pathology. The epithelium of the chorionic villi consists of two layers. The outer is the syncytium, the inner the Langhans layer. Both proliferate in this neoplasm and one or the other cell element may predominate. Chorion epitheliomata are very malignant, disintegrate early, are very vascular, poor in connective tissue and always located in the cavity of the uterus. The growth is villous, soft and of a reddish-blue color. Metastases form very early by way of the blood vessels. The lungs are the most frequent locations for them. The primary tumor

may have entirely disappeared, yet metastases may form. The tumor usually grows very rapidly and causes an early cachexia.

Symptomatology. The cardinal symptoms consist of the known triad, *hemorrhage, discharge* and *pain,* which do not differ from those described under carcinoma uteri.

The *diagnosis* must be based on microscopical examinations of the tumor débris. Bimanual examination elicits a large, soft uterus, and an exploration of the uterine cavity reveals a villous tumor. The history of a hydatid mole is frequently given.

The *prognosis* is bad on account of the early formation of metastases and the usually rapid invasion of the para- and perimetrium.

The *treatment* is, in the absence of metastases, the same as that for carcinoma uteri.

Sarcomata Uteri.

The ratio of the number of carcinomata to sarcomata of the uterus, according to the average frequency of occurrence, is about forty to one. We distinguish two varieties of sarcoma, (1) the mucous membrane, and (2) the wall sarcoma of a. the cervix and b. the body. The first occurs oftener than the second. The corpus and collum are involved with about the same frequency.

Pathology. (1) *Endometrium sarcomata* are usually composed of round and rarely of spindle cells. They are either circumscribed when occurring in the fundus, or diffuse when invading the entire endometrium. They form polypoid tumors which rapidly disintegrate. Hemorrhagic extravasations into the tumor mass are numerous. The surface epithelium and glands resist the invasion of the tumor for a long time, but become finally involved. In the last stages all the tissues of the uterus are invaded.

Cervical mucosa sarcomata are very rare and mostly

spindle-celled. The tendency to polypoid formations is much more marked in the cervical than in the corporeal sarcomata and numerous pedunculated, grape-like masses protrude into the vaginal canal.

(2) The *pathology* of the sarcomata of the myometrium and myocervicum is the same. They occur mostly in myomata. Four per cent. of myomata become sarcomatous. The degeneration begins in the connective tissue of the tumors. The latter rapidly increase in size and disintegrate early. Necrosis, hemorrhages and infections are caused thereby.

Metastases spread through the blood stream and involve distant organs. Sarcomata mostly occur during the fifth or sixth decades, but no age is exempt.

The symptoms of mucous membrane sarcomata are *hemorrhages, serous bloody discharge* with cadaverous odor, and *pains* which are usually labor-like, coincident with the effort of the uterus to expel the tumor.

The constitutional symptoms consist in a rapid loss of weight and strength, anorexia and profound cachexia.

The signs are a large uterus, dilated cervical canal and the protrusion of the tumor through the external os.

The prognosis is bad unless such tumors are seen and treated early.

The diagnosis is based on a microscopical and bimanual examination and inspection.

The *treatment* is the same as for cancer of the uterus.

The symptoms of wall sarcomata are (1) *hemorrhages,* which are more profuse than in myomata uteri, (2) putrid, serous, reddish-colored and profuse *discharges* and (3) *pain,* due to the efforts of the uterus to expel the grape-like masses.

The *constitutional symptoms* consist in a rapid loss of weight and strength, anorexia and marked cachexia.

The *signs* are an enlarged and nodular uterus. An ex-

ploration of the uterine cavity reveals the polypoid, pedunculated masses.

The *diagnosis* is based on microscopical examination assisted by the findings of the bimanual examination and inspection. A rapid growth of myomata must be looked upon with suspicion.

The *prognosis* is bad, unless the sarcoma is recognized early.

The *treatment* is the same as that of carcinoma uteri.

CHAPTER VIII.

FISTULÆ OF THE FEMALE GENITAL CANAL.

Varieties.—Female genital fistulæ are either urinary or fecal. The urinary are (1) vesico-vaginal, including the urethro-vaginal and vesico-cervical, and (2) the uretero-vaginal, including the uretero-uterine and uretero-cervical. The *fecal* are (1) recto-vaginal, and (2) intestino-vaginal, which include the intestino-uterine and the intestino-cervical.

Etiology. Genital fistulæ result from (1) *Injuries* from within sustained during labor, either from a protracted labor or an instrumental delivery. Long-continued pressure of the fetal head on the bladder, ureter and bowels forces these structures against the bony pelvis. A contusion results, causing a necrosis of the organs and the vaginal septum, leading after a few days to sloughing and the formation of a fistula. Trauma resulting from an instrumental delivery may lead to a direct loss of tissue and fistula formation. (2) *Diseases* of the bladder, urethra, ureter, bowels, vagina and uterus, such as vesical stones, severe pelvic infections, ulcerations either tuberculous or cancerous. (3) *Injuries from without* sustained during gynecological operations and accidents, such as the insertion of bougies into the uterus to produce abortion, frequently cause fistulæ. (4) *Foreign* bodies, as pessaries and ligatures accidentally inserted into these structures, also spools, sounds, probes, and other objects used for purposes of masturbation, may cause pressure necrosis or artificial canals.

1. Vesico-vaginal Fistulæ.

Pathology. The fistula may be located median or lateral. The size varies from that of a pin's head to one inch in diameter, or more. The form may be round or irregular. The edges are formed of cicatricial tissue. The mucous membrane of the bladder appears as a bright-red, velvety mass in the lumen of the vagina. Deposits of uric acid salts cause inflammation and incrustation of the vaginal mucous membrane.

Varieties. The most frequent are the vesico-vaginal fistulæ. Other varieties are the urethro-vaginal, the vesico-cervical and the vesico-uterine.

The *symptoms* are an *involuntary escape of urine* through the vagina, a burning and sorensss in the vagina and vulva, and a perceptible urinous odor. If the fistula is located high up in the bladder the urine may escape only if the bladder is quite filled, while the residue passes through the urethra. This usually takes place when the patient is in an upright position, yet when in the recumbent position all of the urine escapes through the vagina. In urethro-vaginal fistula with intact sphincter vesicæ the urine only escapes through the vagina during urination. Chronic cystitis, vaginitis and vulvitis almost always accompany a vesical fistula. The urine undergoes an ammoniacal decomposition in the vagina, causing the characteristic urinous odor of these patients. Pregnancy in the presence of fistulæ is possible, but labor and the puerperium are complicated by infections.

The *diagnosis* is made by inspection, flushing the bladder with colored fluids, cystoscopic examination, urethral catheterization and sounding of the bladder.

The *prognosis* is not bad if the fistulæ are subjected to timely and correct treatment.

Treatment. Prophylactic: In recent injuries distention of the bladder by urine should be prevented by the

insertion of a retention catheter for from five to eight days. While wearing the catheter the amount of urine passed should be recorded every two hours by the nurse in charge. If urine did not pass in the interval time an obstruction of the catheter by urinary deposits is probable. This may be relieved by careful irrigations of the bladder with sterile boric acid solutions through the retention catheter. Should irrigations prove ineffectual, the catheter should be removed, cleansed, sterilized and replaced. To avoid the deposition of urinary salts on the catheter five to ten drops of dilute nitro-hydrochloric acid in a wineglass full of water should be given internally every four hours. If after the final removal of the retention catheter the patient cannot urinate, large amounts of lemonade should be given. Hot stupes should be applied to the supra-pubic region, hot vaginal douches and rectal enemata may also be administered, and finally catheterization must be resorted to. Should the patient complain of incontinence of urine, hydrastis will often be found helpful. For dysuria prescribe hyoscyamus (0.3 c.c. of the tincture t. i. d.), belladonna (0.3 c.c. of the tincture t. i. d.), urotropin (0.5 gm. t. i. d.), potassium or sodium bromid (āā 0.5 t. i. d.). For cystitis use bladder irrigations with 1 : 5000 silver nitrate solutions. If fistulæ form in spite of this treatment healing should be promoted by the application of silver nitrate or tincture of iodin locally, and bladder irrigations conducted with silver nitrate solutions, 1 : 5000. Should the fistula not close within six to eight weeks surgical treatment should be instituted, as a spontaneous closure is improbable after this time.

The *preliminary preparation for operative treatment* is important, as the success of the operation depends largely thereon. The vaginitis and vulvitis should be improved or cured by proper methods, and the amelioration or cure of the cystitis should be brought about by

bladder irrigations. The edges of the fistula should be softened by a gradual dilatation with sounds, Sitz baths, rest in bed and applications of zinc salve to the vaginal mucous membrane. Irrigations of the bladder and vagina will improve the cystitis and vaginitis. Forcible distention of the bladder by closing the fistula temporarily and injecting four to eight ounces of boric acid solution will overcome any existing contraction of the organ.

The *technic* of the operation consists in the exposure of the fistula, the removal of all cicatricial tissue, a flap formation by separating the vaginal from the vesical mucosa, and a splitting of the vaginal mucous membrane transversely in the direction towards which the vaginal flaps can be easiest approximated. The vesical wall is then sutured with catgut, but the vesical surface mucosa must not be included in the suture. In small fistulæ a purse-string suture is sufficient, in large fistulæ interrupted sutures are preferable. The knots must be tied externally to the bladder cavity. An exact coaptation is imperative. Finally the vaginal flaps should be brought in apposition by interrupted catgut sutures which include a bit of the vesical wall to obliterate any dead space between the vaginal and vesical walls. As soon as the operation is finished the bladder should be irrigated to establish its patency. A retention catheter is then inserted into the bladder and the vagina packed with gauze saturated with Beck's paste. The tampon is removed after four days. The after-treatment is the same as described above under the prophylactic treatment. A strict observation of the recumbent position is unnecessary. The patient may leave the bed on the tenth to twelfth day. Sometimes the defect is so large that a transplantation of flaps from the lateral vaginal walls becomes necessary. One of the flaps should remain attached mesially and turn the vaginal surface inward so that the vaginal mucosa forms the mucous coat of the

bladder. The other flap remains attached laterally and is drawn across the site of the fistula in such a manner as to cover it and fill in the vaginal defect made by the turning in of the opposite flap.

Incontinentia Urinæ.

Incontinentia urinæ is treated by a plastic operation on the sphincter vesicæ through the vagina according to Kelley's method.

Uretero-vaginal Fistulæ.

The *symptoms* are the same as those of the vesico-vaginal fistula, except that the urine from one kidney only escapes through the vagina.

The *signs* are the presence of cicatricial tissue at the seat of the fistula. The scar tissue is usually located in the lateral vaginal fornix extending into the base of the parametrium. An intermittent escape of urine may be observed in the region of the vaginal fornix.

Diagnosis. The location of ureteral fistulæ is difficult, but is facilitated by cystoscopic examination (one ureteral orifice does not expel urine into the bladder), by ureteral catheterization and ureteral irrigation and the internal administration of methylene blue.

The treatment aims at the implantation of the upper portion of the affected ureter into the lower portion, or if the latter is too short, a direct implantation into the bladder through an abdominal operation. The best method is Weller van Hook's. Finally if implantation of the ureter should prove a failure removal of the corresponding kidney is indicated.

Intestino-vaginal Fistulæ.

Varieties. Fecal fistulæ are usually *recto-vaginal* and rarely *intestino-vaginal or intestino-uterine.* The size and form vary. The fistula may be large or small, round

or irregular. Secondary changes in the vagina and vulva, as vaginitis, vulvitis with loss of surface epithelium and ulceration, are common and due to contact with intestinal or rectal contents.

The symptoms consist in an escape of *flatus* and *fecal matter* through the vagina, in a recto-vaginal opening, and of *chyme* in intestino-vaginal or intestino-uterine fistula. Flatus and feces escape at irregular intervals, usually after meals. Burning in the vagina and vulva is inevitable.

The *signs* are the presence of intestinal or rectal contents in the vagina. Vaginitis and vulvitis are apparent. The fistulous opening may be exposed by a speculum.

The *diagnosis* is made from the symptoms and the results of the inspection.

The *treatment* is surgical only.

The preparatory treatment tends to ameliorate any existing inflammatory condition by extreme cleanliness and the protection of the vagina with zinc ointment or Lassar's paste.

Operation. Intestino-vaginal fistulæ must be exposed abdominally and the intestinal wall closed in the usual manner. Recto-vaginal fistulæ, which are high up in the vagina, are repaired by the flap-splitting method. If they are low down in the perineal body a complete laceration of the perineal body is produced by an incision including all the structures of the recto-vaginal septum and perineal body beneath the fistulous opening. The edges of the fistula are then refreshened. The vaginal mucous membrane is separated from the rectal mucosa. This separation should extend for quite a distance above the fistulous opening. The rectal mucous membrane is approximated by interrupted chromic catgut sutures, which must include all the layers of the rectal wall except the surface epithelium. The muscle tissue, fascia, vaginal

mucous membrane and perineal skin are coaptated in layers by one continuous catgut suture.

The after-treatment consists in absolute rest. The bowels should be kept quiet for five days, during which time all food should be withheld. Intestinal peristalsis may be checked by codein (0.03 gm. a. m. and p. m.). On the evening of the fifth day an ounce of castor oil is given. As soon as this has acted the patient is given an enema. Soft diet may then be ordered, which after three days is replaced by a light diet. The latter diet is adhered to for at least one week, when general diet may be ordered. Regulation of the bowels to a liquefaction and the administration of a cleansing enema after each bowel movement, should not be overlooked. The patient may be permitted to get up on the seventeenth day following the operation.

CHAPTER IX.

DISEASES OF THE UTERINE TUBES.

Inflammation of the Tubes.

Etiology. (See chapter VI.) The general consideration of inflammations of the genital organs as laid down on page 60 also hold good for similar diseases of the tubes.

Infectious diseases of the tubes are always secondary to a primary infection located in either the lower portion of the genital canal, in the peritoneum and abdominal organs, or a distant part of the body. In other words, the mode of infection is either ascending, descending or hematogenous. Ascending infections of the uterine tubes are the most frequent, and hematogenous the rarest. The inflammatory diseases are acute, subacute and chronic.

Pathology. Pathogenic bacteria may invade the mucous membrane of the tube only when we speak of an endosalpingitis. If they permeate all the layers of the tube they cause a pansalpingitis. Should the tubal abdominal ostium close by an inflammatory reaction or peritoneal adhesions, a sac forms in which the products of the infection remain confined. If the contents are purulent we term the condition a sacto-salpinx purulenta, or pyosalpinx, if serous a sacto-salpinx serosa, or hydrosalpinx, and if hemorrhagic a sacto-salpinx hemorrhagica, or hematosalpinx.

Acute Endosalpingitis.

On macroscopic examination we find the mucous membrane deep-red, hyperemic and edematous, especially at

the fimbriæ. The tubal lumen is filled with an exudate from the blood and lymph vessels. The secretion contains bacteria, serum, mucus and epithelial cells, and is of a clear, watery consistency. It empties into the uterine, and particularly the peritoneal, cavities. The tube is soft and slightly increased in size.

Microscopical examination of the tubal wall reveals an extensive hyperemia and round-cell infiltration of the mucosa, with a destruction of the surface epithelium. The causative pathogenic bacteria may be demonstrated in the mucosa and the exudate. The muscular and serous layers are normal.

A complete cessation of the inflammatory process and a restitution to normal function may occur at this stage of the disease. The invading micro-organisms perish, the secretion is absorbed, the mucous membrane decreases in size and the epithelium regenerates. Such a fortunate outcome of the infection depends on the low degree of virulency and the small number of bacteria, as well as on the reactive powers of the host, which are able to overpower the pathogenic organisms and thus stop the progress of the infection.

Hydrosalpinx.

A continued reinvasion of the causative bacteria, injurious influences, as pronounced hyperemia, caused by menstruation, too frequent cohabitation, and general and local irritations, such as violent physical exertion, obstinate constipation and repeated inconsiderate examinations, cause the infection to persist and increase so that permanent changes occur in the tube. The most important of these is an occlusion of the tubal abdominal ostium due to the increasing thickness of the mucosa. The serous transudate is retained in the tube which enlarges thereby and forms a hydrosalpinx. The tube assumes a retort or posthorn shape. The muscular layer atrophies on ac-

count of the pressure of the retained transudate. The mucosa stretches, which causes obliteration of its folds. The walls become gradually thinner and finally transparent. Microscopically we find in recent cases the changes characteristic of an acute inflammation. Bacteria are present in smear preparations and cultures. In the older, chronic forms the bacteria are absent, the epithelium rests on a connective tissue submucosa, the muscle tissue is atrophic and a few peritoneal adhesions are found at the fimbriæ. Otherwise the serous layer is unchanged. The tube remains freely movable.

The fluid in the hydrosalpinx is clear, thin and serous. It contains albumin, mucin, a few leucocytes and erythrocytes and some epithelial cells.

Hematosalpinx.

An hematosalpinx arises from the rupture of the capillaries of the mucous folds of the tube, due to a marked dilatation of the blood vessels caused by an inflammation or venous stasis. The blood escapes through the ostium abdominale into the peritoneal cavity where it is not absorbed on account of the cell admixture. The blood causes a reactive peritonitis, which closes the tubal ostium and a sactosalpinx is formed. Other causative factors for the formation of a sactosalpinx hemorrhagica are the different atresias of the genital canal, tumor formation of the genitalia and also tubal rupture and abortion during tubal gestation. Torsion of a sactosalpinx serosa or purulenta may also lead to the formation of a hematosalpinx.

Purulent Pansalpingitis.

Purulent infections must invariably cause permanent and irreparable damage to the tube and pass into the chronic stage.

Whether the entire tubal wall may become invaded by

the infective organisms depends on the virulency and number of the organisms and the low resisting power of the host. Microscopically it is seen that the mucous folds are found enormously thickened. The lymph and blood vessels are dilated and contain polymorphonuclear leucocytes, while the tissues are infiltrated with polynuclear pus cells, mononuclear round cells and numerous plasma cells. The latter are recognized by their round or polygonal form, the structure of their nuclei and the characteristic staining properties with methyl-green and pyonin. The muscular and sub-serous layers are edematous and contain islands of round cells which either lead to the formation of an abscess or the development of granulation tissue, and finally of fibrillary connective tissue. This connective tissue is at first rich in nuclear elements, and later, when differentiated, becomes almost devoid of cells. The formation of connective tissue causes atrophy of the muscular tissue.

The serous coat is also thickened and almost entirely loses its surface endothelium. Fibrin is deposited over the denuded areas which leads to agglutinations with the neighboring visceral and parietal peritoneum. In other words, an extensive pelveo-peritonitis always accompanies an acute purulent pansalpingitis. Dense connective tissue bands remain as remnants of the fibrinous agglutinations.

Pyosalpinx.

The tubal abdominal ostium, during the existence of an acute purulent pansalpingitis, closes either by an agglutination of the fimbriæ to each other, or by adhesions between the ostium and the surrounding parietal and visceral peritoneum. The purulent secretion is retained within the tubal sac, a sactosalpinx purulenta has formed. The form of the pyosalpinx is the same as that of a hydrosalpinx. The walls are much increased in size,

due to an active inflammatory proliferation of the epithelium of the mucosa, a connective tissue formation in the musculature and the inflammatory processes going on in the serosum and subserosum. The epithelial metaplasia may lead to a hyperplasia and hypertrophy of the tubular glands, which condition is termed an *adenomatosis*. On the other hand the mucosa may also undergo a complete necrosis and entirely disappear.

The pus contained in the sac may be thin liquid or creamy, or of a yellowish color, and at times have a putrid odor. The causative bacteria are always present in recent infections, while they can be found in culture or smear preparations in about one-half of the chronic cases.

The pus is usually sterile within one year after the occurrence of the infection. The bacteria succumb to their own toxins or die from starvation. An exception must be made in tuberculosis and mixed infections. In the latter virulent bacteria are continuously invading the tubal sac through adhesions with the bowels (colon bacillus).

The *ovary* becomes invaded by continuity in the majority of cases. If a pyosalpinx exists the micro-organisms invade the ovary and abscesses form. If a hydrosalpinx is present chronic cystic oophoritis results. The separating walls between a sactosalpinx and follicular corpus luteum cyst or ovarian abscesses may rupture on account of undue internal pressure, and thus a retort-like cavity forms (a tubo-ovarian abscess or cyst).

Peritoneal adhesions. The visceral and parietal peritoneum becomes rapidly and firmly adherent to an inflamed purulent tube. A rupture of the tubal sac into the bowel, bladder or rectum may take place and a *mixed* infection of the contents of the tube results. Such a complication makes the result of treatment doubtful.

Termination. Pyosalpinx and hydrosalpinx, if left

undisturbed, tend to atrophy during the climacteric. The tubal contents become absorbed, the sac contracts. A presupposition, however, is that the contents of the sac had become sterile through the death of the pyogenic organisms.

Symptoms. Endosalpingitis, sactosalpinx and pansalpingitis have no characteristic pathognomonic symptoms. They are always preceded by the symptoms of an infection of the vagina and uterus, and immediately accompanied by those of peritonitis and oophoritis. The beginning symptoms are those of an endometritis, the accompanying symptoms those of a peritonitis.

Acute salpingitis may cause intense colicky pains and pyrexia, while peritoneal involvement causes severe sensitiveness to pressure, nausea, tympanites and rigidity of the muscles of the abdominal wall.

Chronic salpingitis is usually characterized by pain and abnormal function of neighboring organs, as dysmenorrhea, menorrhagia, irregular menses, sterility and dyspareunia.

The *diagnosis* is made by bimanual palpation, which considers the size, form, consistency, position, mobility and tenderness of the tubes. The question of the virulency or viability of the pathogenic bacteria must also be answered by (1) a leucocyte count and a differential count of the leucocytes (polymorphonuclear neutrophiles must be 75 per cent. or more), (2) careful and correct taking of body temperature and (3) vaccine diagnosis (gonorrhea, tuberculosis).

The *objective signs* are as follows: *In acute salpingitis,* bimanually we find the tube the size of a little finger, movable and firmer in consistency than a normal tube. The fimbriæ impart the sensation of a soft, ill-defined mass. The tube is markedly sensitive.

In *chronic pansalpingitis* the tube is irregular, convoluted and the size of a thumb. The consistency is firmer

than normal, the range of mobility appears restricted on account of adhesions. The upper half of the tube is found laterally to, and the lower half behind, the uterus. The latter may be drawn toward the affected side. There is marked sensitiveness on palpation. In salpingitis isthmica nodosa nodular swellings may be palpated.

Hydrosalpinx, hematosalpinx. The tube is outlined as a retort-shaped mass, is tense, elastic, fluctuating and often adherent at the fimbriæ. The ovary can rarely be recognized apart from the distended tube.

Pyosalpinx. The uterus is restricted in mobility and displaced. The tubes are located laterally and behind the uterus and are directed downward. The consistency is firm at the uterine insertion and soft at the fimbriæ. The shape is retort-like. The size of a sactosalpinx may increase up to that of a lemon. The tubes are limited in range of mobility and are very sensitive on palpation and to pressure. The ovary can be palpated apart from the tube only in exceptional cases. They may have formed a tubo-ovarian tumor. With retro-flexion the tubes are found in Douglas's pouch, with ante-flexion laterally and posteriorly to the uterus.

A *differential diagnosis* must be made between salpingitis and (1) extra-uterine pregnancy, (a) before rupture and (b) at time of rupture, (2) appendicitis, (3) sigmoiditis, (4) tumors of the ovaries, (5) tumors of the uterus, (6) parametritis and hematoma, and (7) perimetritis and hematocele. (See table 5.)

The *prognosis* of salpingitis is usually favorable as regards life, but doubtful as to restoration of health and the conservation of sexual function. Loss of earning capacity and enjoyment of life, together with sterility, are the frequent results of the severer infections. The prognosis in puerperal and gonorrheal infections is usually favorable, compared with that of tuberculous infec-

	tumor.	Para- and perimetritis.	Hematoma and hematocele.
History.	No menturbances.	Infection, trauma, as labor, operation.	Tubal pregnancy or trauma as operation, labor.
Symptoms. Onset.		Sudden.	Sudden.
Rigor.		Marked.	Normal.
Pyrexia.		Yes. 101 to 103.	Moderate 100 to 101.
Pain.		Continuous, severe.	Continuous.
Pulse.		Rapid, sharp.	Negative.
Temperature.		Constant or irregular.	Due to absorption.
Symptoms refers pelvic organs, of lesion.	ain. e with n and def-	Peritoneal irritation with nausea, vomiting and constipation.	None except fullness in pelvis and peritoneal irritation.
Abdomen inspecti	as only ed tumors sidered main usu- pelvic.	Distended. Rigid.	Distension in hypogastrium.
Percussion.		Tympanites.	Dullness over bloodtumor.
Palpation.		General.	Sensitiveness over bloodtumor. Marked.
McBurney's point		Negative.	Negative.
Morris' point.		Tender.	Negative.
Uterus. Position.		Adherent to mass.	Enclosed in blood mass or lateral to it.
Movability.		Impeded, fixed.	Impeded.
Size.			
Form.		Depend on fact whether metritis or endometritis coexist.	Normal.
Consistency.			Normal.
Sensitiveness.			Normal.
			Normal.
Adnexa. Positio	except en- ary due to mation.	Imbedded in exudate and not palpable.	Enclosed in blood mass, and usually not palpable.
Movability.			
Size.			
Form.			
Consistency.			
Sensitiveness.			
Mass. Location.	osterior to retro- ouch.	Lateral to uterus in parametritis. Post. in perimetritis tends to diminish with progressing organization.	Lateral in hematoma and posterior in hematocele tends to diminish with progressing organization.
Vaginal fornices.		Bulging lat. or post.	Bulging laterally or posteriorly.
Hemanalysis.		Negative.	Negative. Perhaps secondary anemia.
Red blood corpu		Negative.	
Percentage of globin.		Increased.	
Leucocytes.		75 per cent or more.	
Neutrophiles.		Negative.	Negative, excepting pregnancy.
Abderhalden.			

tions, which are unfavorable. Chrobak claims that normal labors occur in about 16 per cent. of unilateral infections, and in 7 per cent. of bilateral disease.

Treatment. Prophylactic. Treat infections of the external genitalia immediately, and especially prohibit coitus, unnecessary examinations and instrumentation during the acute and subacute stages.

Medical. For the acute stage: Abstain from local treatment and frequent examinations during the acute stage, and considerably beyond it. Interdict sexual congress or excitation, which may cause an increased congestion in the pelvic organs and thereby an exacerbation of the condition. Absolute rest in bed in Fowler's position, the application of an icebag or a Leiter coil, or hot fomentations, to the abdomen should be advised. For the pain prescribe aspirin (0.5 every four hours), and, if necessary, morphine. For profuse and burning leucorrhea advise vaginal douches of normal saline solution at 115° F., under low pressure. Food should be omitted when peritoneal involvement is present. Constipation is relieved by enemas during the acute stages, and later on by saline cathartics.

For the sub-acute stage, i. e., after the patient has been free from fever for at least four weeks, use the hyperemic treatment of Bier, phototherapy, Sitz baths at 90 degrees F., to which half a litre of infusion of oak bark may be added, hot vaginal irrigations at 120 degrees F., Priessnitz compresses to the abdomen during the night, with easily digestible, nourishing diet, rest in bed and exercise in the open air. Locally, to stimulate absorption, employ tamponing, iodin douches, and scarifications (only, however, in the absence of virulent germs). Regulation of the bowels is imperative. Finally, change of surroundings should be insisted on; mud baths are very beneficial. Massage is contraindicated during this stage. The fore-

going treatment should be immediately discontinued if a rise in the temperature occurs.

About sixty-five per cent. of the patients may be permanently relieved by this treatment and enabled to enjoy life and resume the duties of their daily routine work. An additional twenty to twenty-five per cent. may be able to work, but have slight subjective symptoms. While in the remaining ten to fifteen per cent. recourse should be had to surgical treatment. The primary mortality of the latter is about five to ten per cent. The percentage of cures is about sixty in conservative, and eighty in radical, operations.

Surgical treatment is indicated in the *acute stage* if the infection causes (1) *danger to life,* as shown by a constantly increasing pulse rate, loss of strength and abscess formation; and (2) when there are signs of a *general peritonitis;* and in the *chronic stage* when (1) the above mentioned directions for medical treatment have been faithfully but *unsuccessfully* carried out; and if repeated *recurrences* and exacerbations occur and the patient must be saved from continued ill-health and loss of earning capacity, as evidenced by pain, invalidism, menorrhagia and dyspareunia; and (3) when *sterility* exists which is accompanied or not by subjective symptoms.

The *operative procedures* consist, in the acute cases, in an incision and drainage of the abscess through the posterior vaginal fornix. The risk of the operation is slight and the procedure is often followed by a complete recovery. In *general peritonitis* an *abdominal section with drainage* is the procedure of choice. The after-treatment consists in starvation, Fowler's position and continuous proctoclysis. In *chronic cases* we advise abdominal section with either *salpingostomy* or *salpingectomy.* Salpingostomy must be performed if tubes and ovaries are to be saved and the patient desires offspring.

Extrauterine Pregnancy.

Synonym. Ectopic gestation or pregnancy.

Definition. Extrauterine pregnancy means the arrest and development of a fertilized ovum either in the uterine tube, ovary or peritoneum.

The *varieties,* according to the location, are tubal, ovarian, and peritoneal. The tubal is the most common, the two latter locations are very rare.

Tubal Pregnancy.

We understand by tubal pregnancy the implantation of an impregnated ovum in the tube instead of in the uterus. There are *three varieties* of tubal implantation, according to location in the tube. The *ampullary* implantation is the most, the *isthmic* the next, and the *interstitial* the least common.

Pathology. Fecundation of a human ovum occurs in the ampulla, i. e., each pregnancy begins extrauterine and the possibility of a tubal pregnancy is thus always present. Segmentation and development of the impregnanted ovum commence at once and when the embryo is retarded in its onward travel, the ovum by the eroding action of the trophoblast, with which it is surrounded, becomes imbedded in the tubal mucosa. The imbedding may occur either in the space between two tubal folds, *intercolumnar,* or on the apex of a fold, *columnar.* A decidua, in which the ovum might develop as in the uterus, is not present in the tube and the ovum immediately grows into the connective tissue and musculature of the tubal walls. The chorionic villi with the cell layer of Langhans advance into the tubal walls and soon afterwards the ovum is situated *externally* to the lumen of the tube. The latter becomes either entirely obstructed at this point, or remains patent, depending on the development of the ovum.

The uterine tube is not capable of maintaining growth

with the ovum. The columns of Langhans' cells of the chorionic villi, which form the periphery of the ovum, proliferate through the musculature into the serosa. During this proliferation the blood vessels of the tube do not usually remain entirely intact, their walls become eroded and *hemorrhages* occur whereby the growth of the ovum is, as a rule, interrupted. The blood forms a *hematoma mole* within the ovum. If the tubal wall at the periphery of the ovum becomes completely eroded, the tube ruptures. If the rupture occurs towards the lumen of the tube, we speak of an *internal rupture of the fetal sac* or a *tubal abortion,* while if the sac breaks through the tubal wall, we speak of an *external rupture* of the fetal sac, or *tubal rupture.* Tubal abortions are more frequent than tubal ruptures. The former usually occur in ampullary, and the latter in isthmic, pregnancies.

With the occurrence of a rupture of the fetal sac the growth of the ovum is interrupted. Exceptionally, the placenta may become engrafted on the peritoneum, whence a new blood supply is provided and the ovum continues to grow.

Tubal rupture and abortion are always accompanied by intra-abdominal hemorrhages. These are usually more profuse in tubal rupture than tubal abortion, but in both may become dangerous to the life of the patient. The escaped blood collects, according to the law of gravity, at the lowest point of the abdominal cavity and forms a retro-uterine hematocele. If the rupture of the tube takes place between the folds of the broad ligament, then the blood escapes into the tissue underneath the peritoneum and an extra-peritoneal hematoma forms.

Smaller hematoceles are gradually and completely absorbed. Larger ones become encapsulated. A reactive peritonitis sends connective tissue bands into the mass, dividing it into several parts. The blood is finally absorbed, the connective tissue bands form adhesions be-

tween the pelvic organs and bowels and a *chronic adhesive pelveo-peritonitis* remains. This process may drag along for years.

A hematocele may become *infected* either by way of the tube or from adherent intestinal loops. A pelvic abscess forms, which in turn may rupture into the bowel, bladder, vagina or through the abdominal wall.

Tubal pregnancy, however, may terminate in another way. The fetus, within or without the fetal membranes, remains connected with the placenta, which becomes adherent to the peritoneum and thus secures its blood supply. The fetus thus continues to develop in the free peritoneal cavity, where it may perish at any time and, manifestly, always at full term unless it is then delivered through a cœliotomy. Should the fetus die and for one reason or another be left within the abdominal cavity its fate becomes variable. If the tubal pregnancy is interrupted in its early stages, the fetus may then become completely absorbed. If the development of the fetus has advanced to bone formation, then the soft parts are absorbed and the skeleton remains, or the fetus becomes imbedded in connective tissue, dries up and mummifies. If the connective tissue capsule is impregnated with lime salts a lithokelyphos forms; if the calcification also invades the superficial layers of the fetus, a lithokelyphopedion results, and if the entire fetus calcifies, then a genuine lithopedion is produced. Infection of the fetal sac may also occur leading to suppuration of the soft parts of the fetus.

The uterus participates during the first two months of the extrauterine pregnancy by enlargement, softening, engorgement and the formation of a decidua. The decidua, as a rule, is expelled with hemorrhage and labor-like pains at about the eighth week. The expulsion of the decidua is followed by a regeneration of the endometrium.

Etiology. It is thought that tubal pregnancy is caused

by (1) anomalies of the ovum (follicular epithelium, germinating disc), (2) infantilism of the tubes (convoluted tubes), (3) anti-peristalsis of the tube and absence of ciliary motion of the surface epithelium, (4) polypi in the tubes obstructing its lumen, and (5) old chronic inflammation of the tubes interfering with the passage of the ovum. The latter is the most probable theory.

The *symptoms* should be considered under two divisions: (a) if pregnancy is not interrupted and (b) if pregnancy is interrupted.

A. The patient suspects pregnancy and experiences amenorrhea, a feeling of warmth in the pelvis and fullness in the abdomen, a sensation of swelling and drawing of the breasts, nausea, morning-sickness, disturbances of the functions of the stomach, and, lastly, undefinable pains and discomfort until the time of quickening, when fetal movements are felt. At full term, spurious labor occurs characterized by colicky, bearing-down labor pains in the pelvis, expulsion of the uterine decidua and lochia. Death of the fetus takes place if no surgical interference is offered, being indicated by a cessation of labor pains and ill-health of the mother, manifested by rigors and general malaise.

If suppuration occurs in the fetus and sac, the usual symptoms of infection and abscess formation are present. Calcification or mummification of the fetus causes severe disturbances of the general state of health, although unusual symptoms may not occur. Local symptoms appear only when the calcified or mummified fetus presses on neighboring organs, or interferes with physical exercise, or delays a subsequent labor of the carrier. Women, however, may become pregnant, intra-uterine, after the death of an extra-uterine ovum. Lithopedions are often accidental findings during future ante or post mortem examinations.

B. The symptoms of tubal rupture and abortion dif-

fer, depending (1) on the time of gestation, (2) on the amount of blood lost, resulting in either the formation of an hematosalpinx, hematoma or hematocele, with the possibility of the occurrence of an infection in these, and (3) on the fate of the ovum, whether it continues to develop, whether calcification or mummification occur, or infection takes place. The ovum ordinarily perishes in the first two weeks of extra-uterine pregnancy, as a result of the hemorrhage, without subjective symptoms caused thereby. The ovum, the size of a cherry or plum stone, may remain in the tube and be accidentally found during a subsequent operation for another pathological condition. If we then incise the tubal walls, remove the ovum and close the incision by suture, involution of the organ results with complete preservation of the function of this organ. Tubal abortion may lead to the formation of an hematosalpinx, hematocele, or hematoma. Tubal rupture always results in an hematocele or hematoma. Interruption of tubal gestation usually takes place during the first three months of pregnancy, if the ovum did not perish during the first two weeks.

An *hematosalpinx formation* causes drawing pains in the deep lateral portion of the pelvis and shock. If infection occurs therein a pyosalpinx results. The infection causes pyrexia, pain, tympanites, urinary and rectal irritation. If the abscess ruptures, the contents of the sac (pus, fetus, débris of the ovum) are expelled, thus terminating either in a complete recovery or in the formation of a permanent fistula.

The formation of an *hematocele* is accompanied either by slight or profuse hemorrhage. The slight hemorrhage is accompanied by some pain and shock. The blood is afterwards readily absorbed and the patient recovers completely. Should the hematocele cause a peritoneal irritation, then peritoneal adhesions and chronic pelveo-peritonitis result. However, if the

amount of blood lost is large, one severe, labor-like pain may take place, with immediate and profound shock. If help is not immediately rendered the patient may succumb suddenly. On the other hand, an abatement of the symptoms and arrest of hemorrhage may follow. This may be either temporary or permanent. If temporary another hemorrhage may occur which then may cause death of the patient.

The uterine decidua at the time of the tubal abortion or rupture is expelled through the uterine os with labor-like pains and hemorrhages. Infection of an hematocele may occur, resulting in a pelvic abscess. A hematoma presents the same symptoms as an hematocele. Shock and amount of blood lost are usually less severe than in hematocele.

The ovum after being detached from the tube may become immediately connected to its parent tissue and develop to its physiological maturity. The fate of the fetus under these circumstances has been above described.

Diagnosis and signs. The diagnosis of an uninterrupted extra-uterine pregnancy depends on the history and a bimanual examination, which must establish the following facts: 1. Palpation of the uterus and exclusion of an intra-uterine pregnancy. 2. Demonstration of the typical condition of the pregnant tube. The tube lies laterally and posteriorly to the uterus, is drumstick-shaped, its largest circumference corresponding to the seat of the ovum. It is of a soft consistency, freely movable and is not sensitive to pressure.

If a decidua should be passed by way of the vagina, or quickening occur, or the fetal heart beat is perceptible and the mass lies external to the uterus, the latter not corresponding in size to the time of gestation, then the diagnosis is not so difficult.

The *diagnosis of external rupture with large hematocele formation,* is based on (1) sudden, violent, labor-like

	vidi a.	Myomata uteri up to size of fist.	Ovarian tumors up to size of fist.
History.		Gradually increasing menorrhagia. Sterility.	Negative.
Onset.	np-	Insidious.	Insidious.
Symptoms.	to Is- ra-	Abnormal menstruation. Leucorrhea. Dysmenorrhea. Pressure-pain.	Negative, except weight; limitation of space. Usually sterility.
Pulse.		Normal, especially myoma heart.	Normal.
Temperature.		Normal.	Normal.
Uterus. Signs.			
Position.		Normal, depending on seat of tumor.	Depends on size of tumor.
Movability.			Depends on size of tumor.
Size.	ing	Depending on size of tumor.	Normal.
	to	Increased.	
Form.		Irregular.	Normal.
Consistency.		Hard, firm.	Normal.
Sensitiveness.	ion.	None.	Negative.
Adnexa.			
Position.		Normal, depending on size and location of tumor.	Depends on size of tumor.
Movability.			Depends on size and location of tumor.
Size.		Normal.	Ovary increased in size.
Form.		Normal.	Symmetrical or irregular.
Consistency.		Normal.	Hard and firm.
Sensitiveness.		Normal.	None.
Location of m	on- ver-	Included in any part of the uterus.	Usually posterior to uterus.
Blood.			
Reds.		Secondary anemia.	Normal.
Whites.			Normal.
Hemoglobin.		Normal.	Normal.
Neutrophiles.		Normal.	Normal.
Abderhalden.		Negative.	Negative.

pains of extreme intensity, (2) shock and syncope, accompanied by nausea, vomiting and peritoneal irritation. The latter is indicated by tenesmus, rigidity and sensitiveness of the abdominal wall; (3) progressive anemia with *decrease* in temperature of the body and *increase* in the pulse rate (pulse becomes feeble, wiry, finally irregular and imperceptible), (4) flatness on percussion over the lower abdomen, i. e., over the blood mass and above this mass tympany, (5) on bimanual examination the uterus is found softened, increased in size and surrounded by a fluctuating mass located laterally and behind the uterus, and in extreme cases also anteriorly and superiorly, when it bulges into the vagina. As soon as coagulation is complete the mass becomes hard. The adnexa and fetus are only exceptionally palpable. Finally Abderhalden's serum diagnosis and a hemanalysis will give valuable aid.

The differential diagnosis must be made between extrauterine pregnancy before and at the time of rupture and uterine pregnancy, retro-flexio uteri gravidi, salpingitis, appendicitis, uterine and ovarian tumors and ruptured pyosalpinx. (See Table 6.)

Hematocele requires a differentiation from pelveoperitonitis; hematoma from pelvic cellulitis. (See the respective chapters.)

The *prognosis* is grave under all conditions. It becomes favorable if the tubal pregnancy is recognized before the occurrence of rupture and the products of the extrauterine pregnancy are surgically removed.

The *treatment* is surgical prior to rupture, as well as during or soon after rupture. If the patient is not seen at this time and the fetus continues to develop after a rupture and signs of quickening are present, the treatment should be expectant. Even if rupture occurs and the patient is moribund, with the pulse imperceptible but respirations still present, an operation should be per-

formed to arrest the hemorrhage. Shock is combated by hypodermoclysis or transfusion either of blood or normal salt solution, by bandaging the extremities, by raising the foot of the bed, by the application of heat and the administration of stimulants.

If the patient comes under observation after the hematocele has existed for a long time and organization taken place, and if absorption causes ill-health, or if suppuration ensues, we should perform a posterior colpotomy (with T-rubber drainage). For all other conditions the operation should be performed through an abdominal incision.

If the salpingocyesis is still intact, the ovum may be removed by salpingectomy or salpingostomy.

In operations on advanced cases of extra-uterine pregnancy, the removal of the sac and placenta may cause difficulty. The rules to be observed are as follows: Up to the end of the fourth month, no objection exists to a total removal of the sac. After that time the case should be watched. At full term, i. e., during the last month of pregnancy, the fetus should be delivered by an operation if it is still alive. In all other conditions it is better to wait two months to allow the placenta to atrophy, unless sepsis calls for an earlier removal.

The gestation sac in the later stages of tubal pregnancy is usually adherent to the intestines and omentum. If the adhesions can be safely broken up, the sac may be removed *in toto*. If not, the edges of the sac should be stitched to the abdominal wound and the sac packed with iodoform gauze. The rules for the treatment of the placenta are: (1) When the placenta is situated above the fetus, attempt removal. (2) When below the fetus, leave it. (3) The placenta may become detached during the operation, then ligate the uterine and ovarian vessels and all oozing surfaces. (4) If the placenta should be left behind, sequester it by closing the aperture in the peri-

toneum, beneath the placenta, close the sac and transfix the latter to the peritoneal incision. If suppuration should occur, drain. (5) If the placenta is atrophied it can be removed with little fear of hemorrhage. (6) After all it is a wise plan to remove the placenta at the primary operation. Remember Momburg's method of producing pelvic ischemia at all periods of extra-uterine gestation and during operations for the removal of the gestation sac.

CHAPTER X.

DISEASES OF THE OVARY.

1. Deformities of the Ovaries.

These are congenital and not of frequent occurrence.

Bilateral aplasia or absence of both ovaries is found with total aplasia of the tubes and uterus; *unilateral aplasia* with aplasia of the corresponding portion of Müller's duct. *Hypoplasia* occurs with hypoplasia of the corresponding portion of Müller's duct; a pseudo-hermaphrodism of the external genitalia is usually associated with this condition.

Infantilism of the ovary is indicated by its peculiar shape and anomalous position. It is elongated and lies above the brim of the pelvis just beneath the kidney. Its axis is parallel to that of the spinal column. An infantile condition of the ovary does not have any influence on its function.

Supernumerary ovaries should be differentiated from those where the ovary has become divided as a result of an inflammatory pseudo-membrane or a torsion. Accessory ovaries are of importance, as tumor formation may occur in them or pregnancy result after supposed castration operations.

Hypertrophy and atrophy of otherwise normal ovaries may occur without any demonstrable cause.

The diagnosis of all these congenital conditions may be very difficult.

Symptoms are not manifest until the commencement of

menstrual life. An exploratory laparotomy may be justifiable to clear up the diagnosis.

2. Ovarian Displacements.

Displacements or prolapses are usually secondary to those of the uterus (especially retro-flexio uteri), and to extensive relaxations of the pelvic connective tissue.

Symptoms and signs. The ovary lies on the floor of the recto-uterine pouch, where it is exposed either to trauma during coitus (dyspareunia) or to pressure during the expulsion of constipated stools (tormina-tenesmus). It finally becomes very sensitive. Urination is frequent and painful (dysuria). As a result of these injurious influences the maturing of a follicle, the pressure of the bowels during the upright position, or physical exercise cause pain.

The *diagnosis* is made from the objective signs and subjective symptoms. The ovary has the characteristic form, is increased in size, occupies an abnormal position and is very sensitive. A differentiation from floating kidney (nephroptosis) or prolapsed spleen (splenoptosis) should be made. The palpation of these organs in their normal places decides the diagnosis.

Treatment. Replacement of the uterus and the insertion of a pessary also replaces a prolapsed ovary. Vaginal douches of an infusion of conium maculatum (two or three tablespoonfuls of the powdered leaves in two quarts of water) or suppositories of morphine, opium or codeine may be given for pain. Regulation of the bowels so as to liquefy their contents is indicated. Sexual congress must be interdicted. If these measures fail to give relief ovariopexy should be performed by shortening the utero-ovarian ligament, or Webster's endo-peritoneal shortening of the round ligaments may be executed, which also lifts the ovary into its normal position.

Ovarian Hernia.

Oophorocele is mostly inguinal, rarely femoral. It is usually congenital and bilateral. The uterus is lateroposed towards the side of the hernia. The tube, as a rule, also passes through the canal into the sac (salpingo-oophorocele).

Symptoms. Increase in the size of the ovary and sensitiveness of the contents of the hernial sac during menstruation are characteristic. Hemorrhages and inflammatory tumor formation occur as a result of the exposure of the organ to external injuries. The ovary is the size of a plum or egg (hydrops of hemorrhagic follicles may increase its size), and is always sensitive.

Diagnosis. The signs of inguinal hernia are present (swelling, impulse on coughing, reducibility). An altering sensitiveness and an increase in size of the content of the hernial sac corresponding to ovulation may be observed, also a deviation of the uterus towards the seat of the hernia. In femoral oophorocele we encounter the same signs, but the sac is located beneath Poupart's ligament and the inguinal canal is free. Ischiadic, abdominal and obtuator hernias should also be thought of.

The *treatment* consists in a herniotomy and a replacement of the ovary. Non-surgical reposition is impracticable.

3. Circulatory Disturbances.

Congestion may be physiological during the pre-menstrual stage, or pathological when caused by torsion of the ovarian ligaments, which occurs especially during a displacement or tumor formation of the uterus. Cardiac, renal or hepatic stases may also cause ovarian congestion.

Pathology. The rupture of blood vessels causes hemorrhage into the ovary, with destruction of tissue leading to the formation of apoplectic foci in the ovarian tissue.

Follicular Hematoma.

The *direct causes* are physical exertion, venereal excesses and bimanual examinations, especially at the time of menstruation. A predisposition arises from a hyaline degeneration of the vessel walls in the ovary during the involution of a corpus luteum. Complete absorption of the hematoma is usual. If it fails to take place then the hematoma causes pressure symptoms. Suppuration also may be observed, resulting in a pelveo-peritonitis. Interstitial hematomata are rare and occur during severe sepsis.

Symptoms are not always caused. They occur if the hematoma is of a large size and absorption retarded, and consist of a burning pain localized in the ovary. Rupture of a follicular hematoma is accompanied by sharp pains due to the peritoneal irritation, with collapse and the formation of a hematocele.

The *diagnosis* is made from the sudden onset, the pain, the enlargement of the ovary and the rapid resolution.

The *treatment* consists in absolute rest and the application of an ice bag or ice coil to the abdomen. Absorption is stimulated by the use of ichthyol tampons and iodine douches. In urgent cases with profuse hemorrhage resection of the diseased portion of the ovary is preferable.

4. Inflammations.

Oophoritis is almost always secondary to salpingitis. But the exciting agent may also invade the ovary from the abdominal cavity through the intestines and from the blood and lymph streams.

We distinguish acute and chronic inflammations. The latter are, as a rule, the result of the former.

Etiology. Oophoritis is in the majority of cases infectious. In a few cases it may result from chronic circula-

tory disturbances. The bacteria usually concerned are: (1) Pyococci, (2) gonococci, (3) tubercle bacilli, (4) colon bacilli and, rarer, (5) pneumococci, (6) typhoid bacilli, (7) actinomyces and (8) spirochetæ pallidæ.

A. Acute Oophoritis.

Pathology. The infectious agent reaches the ovary (1) ascending either by continuity from the tubes or through the lymph channels of the ligamentum latum, or by thrombosed veins through the hilus ovarii; (2) descending through adhesions from the bowels, or (3) hematogenous through the blood or lymph stream from distant foci. The ovary becomes covered by a thick, fibrous and purulent exudate. On section marked hyperemia, edema, and multiple abscesses are seen. Microscopically round-cell infiltration of the entire ovary, purulent softening and destruction of the follicles, are observed. Bacteria can be demonstrated in the blood and lymph vessels. If a corpus luteum exists, a corpus luteum abscess forms. Gonorrheal infection of the ovary always ascends from the tube and causes a peri-oophoritis as the gonococcus cannot penetrate the firm connective tissue albuginea, unless a follicle ruptures, when it forms a corpus luteum abscess.

B. Chronic Oophoritis.

Acute oophoritis may heal completely. The bacteria are overpowered by the organism, the edema is absorbed, the abscesses inspissate and undergo connective tissue degeneration and the fibro-purulent deposits become organized and form adhesions with the neighboring organs. However, recovery may be incomplete. The abscesses remain. The ovary and tube become intimately adherent to each other. The ovarian abscess cavities coalesce with the tubal abscess and form a tubo-ovarian abscess. The bacteria contained in the sac may either perish or retain

their virulence for a long time. Mixed infections may also arise from a migration of intestinal bacteria through the adhesions into the tubo-ovarian abscess (bacillus coli and, more rarely, actinomyces). Ovarian abscesses may completely disappear during the climacteric, provided the bacteria do not retain their virulence. Deposits of lime in such an ovary may take place, resulting in the formation of an ovarian stone.

The infection, however, may not always lead to abscess formation. It then causes a chronic connective tissue proliferation, a *chronic interstitial oophoritis*. The ovarian stroma becomes firm and poorer in nuclei. The vessels undergo a connective tissue metaplasia. The primary follicles gradually disappear. Finally the albuginea participates in the connective tissue proliferation, and rupture of a Graafian follicle is prevented. The ovum perishes and a follicular cyst remains. The latter may attain the size of a child's head and is lined with a layer, or layers, of cubical epithelium. Such ovaries are, finally, interspersed by quite a number of larger and smaller follicular cysts (small cystic degeneration of the ovary). The septa between small cysts may burst, leading to the formation of larger cysts. Such cysts may be lined with a lutein membrane and are then known as corpus luteum cysts. The contents resemble a follicular cyst. A corpus luteum cyst must be differentiated from a corpus luteum hematoma, caused by stasis. Small cystic ovaries may ultimately communicate with an existing hydro-salpinx and form a tubo-ovarian cyst.

Chronic hyperemia of the ovary caused by myomata, displacement, cardiac diseases, nephritis and hepatitis, venereal excess and masturbation may cause a chronic oophoritis with follicular cysts. However, the possibility of the existence of a previous infection must always be considered.

The symptoms of an acute oophoritis are masked by

those of the inflammation of surrounding tissues. With abscess formation we usually find symptoms of pus infection. If organization and resolution of pelvic infections take place, the ovary shows a tendency to lag behind in this process.

The diagnosis is made bimanually. The combined examination elicits enlargement, sensitiveness and immobility of the ovary.

The treatment is the same as that of any other acute pelvic infection.

The *symptoms* of chronic oophoritis are pain, dysmenorrhea, menorrhagia, intestinal disturbances and usually also sterility. The pain is pronounced, burning and constant and aggravated by every exertion. Menstruation is painful, frequently irregular, and always abundant. Constipation and gastric disturbances are usually present. The pressure of hard fecal masses on the ovaries increases the ovarian pain. Sterility is the rule.

Diagnosis. A sensitiveness and enlargement of the ovary are elicited on bimanual examination. The organ is immovable, irregular in outline and adherent to the surrounding structures.

The differential diagnosis must be made from tumors of the ovary. Tumors are not sensitive, continue to enlarge and there is an absence of menstrual disturbances.

Treatment. The indications for treatment of chronic oophoritis are continued pains, ill-health and sterility. If the inflammation is subacute we order rest in bed, sexual abstinence, regular evacuations of the bowels and ice bag or Priessnitz compresses for pain and sensitiveness. This course is followed by iodine douches, tamponing and saline or mud-baths to promote resolution. Bier's hyperemic method in the form of dry heat is also good treatment. Change of surroundings and climate

is often beneficial. Surgical treatment is indicated by continued ill-health and loss of working and earning capacity after medical treatment has failed. It consists in a resection of the diseased portions of the ovary, especially of the cystic degenerated portions, or puncturing the small follicular retention cysts, and eventually an oophorectomy.

Ovarian Tumors.

General remarks. Neoplasms of the ovary are, next to the uterine, the most frequent of genital tumors. They are the most common of all intra-abdominal tumors in the female.

The *etiology*, with the exception of the embryonal tumors, is unknown.

We distinguish the following varieties:

I. Epithelial-cell tumors:
   1. Cystoma glandulare multiloculare pseudomucinosum.
   2. Cystoma serosum glandulare papillare.
   3. Carcinoma:
      (a) primary ovarian carcinomata,
      (b) malignant degeneration of cysts and embromata,
      (c) metastastic ovarian carcinomata.

II. Connective tissue tumors:
   1. Fibromata, also pedunculated fibroids.
   2. Fibromyomata.
   3. Sarcomata.

III. Ovigenous or embryonal tumors.
   1. Cystic embryomata or dermoids.
   2. Solid embryomata or teratomata.

I. Epithelial Cell Ovarian Tumors.

These originate from the germinal epithelium. This may undergo metamorphosis into ciliated epithelium.

Hence we have non-ciliated and ciliated epithelial ovarian tumors. The non-ciliated epithelium secretes a stringy fluid containing pseudo-mucin and forms pseudo-mucinous cysts. The ciliated secretes a thin, liquid, serous fluid and forms serous cysts.

1. Cystoma Glandulare Multiloculare Pseudomucinosum. Pseudomucinous Ovarian Cyst.

Pathology. By a proliferation of the germinal epithelium gland-like tubules are formed which penetrate the ovarian stroma in all directions. The neck of these glandular tubes becomes constricted by a reactive proliferation of the ovarian connective tissue and retention cysts are formed. The germinal epithelium in a given cyst continues to proliferate and multiply and form secondary or daughter cysts. This process of proliferation and cyst formation is unlimited. The individual cysts grow, due to a continuous profuse secretion and epithelial proliferation which results in a conglomeration of large and small cysts, a cystoma glandulare multiloculare. The connective tissue found in the cysts is derived from the ovarian connective tissue. It also participates in the proliferation to form the supporting structure or skeleton of the cyst. However, its growth cannot keep pace with that of the epithelial proliferation and continued secretion of the cystic fluid. It becomes stretched out to a very thin tissue on account of the gradually increasing internal pressure and finally atrophies. The atrophied partition wall between two cysts breaks down and a coalescence of several cysts into a larger cyst takes place. Connective tissue bands remain as remnants of the original partition walls, proving the development from multiple smaller cysts. Apparently solid tumor masses are found in such multilocular cysts. But on microscopical examination it can be shown that these masses are composed of innumerable smaller cysts.

Microscopically it is seen that the cyst walls are lined with a single layer of high-bodied cylindrical epithelium, the nuclei being placed at the base of the cell. Each cell represents a highly glossy, protoplasmic body. It may become flattened to a cubical form by a high internal pressure. An *atypical proliferation* of the epithelium or a *polymorphism* or *multiplication of the epithelial cell layers* suggesting malignancy are never seen. Papillomatous elevations may form in the interior of the cyst cavities. They consist of a connective tissue basis covered with a single layer of the described typical epithelium.

The contents of such a cyst is a colorless, glossy, sticky, stringy fluid, rich in albumin. It contains pseudo-mucin as the characteristic chemical base. The color of the fluid may change to a chocolate-brown, red, yellow, or green, due to an admixture of blood coloring matter. The different colors correspond to the stage of decomposition of the blood. The hemorrhage may result from erosions in the cyst walls or a torsion of the pedicle of the tumor.

The pseudo-mucinous multilocular glandular ovarian cysts may attain an enormous size, fill out the entire abdomen, pushing up the diaphragm and causing dyspnea and even death by suffocation.

The form may be similar to that of an ovary, or it may present grooves and bosses corresponding to the different individual cysts. The surface is smooth and glistening.

A spontaneous rupture of a cyst may result from increased internal pressure. The contents, which then enter the abdominal cavity, are either absorbed, or if viable epithelia escaped, the latter may become grafted upon the peritoneum, proliferate and secrete. They become encapsulated by a reactive peritonitis and a *pseudomyxoma peritonei* results. Dangerous disturbances may ensue from the pressure of the tumor on blood vessels,

especially the portal vein. These cysts are usually unilateral, but may be bilateral. If such a cyst be removed a similar formation may occur, often after many years, in the other ovary. The cysts also may undergo carcinomatous degeneration.

2. Cystadenoma Serosum Papillare.

The germinal epithelium proliferates, undergoes metamorphosis into ciliated epithelium and a serous ovarian cyst is formed. It is a multilocular cyst like the pseudomucinous and has the same structural characteristics. The distinguishing difference of a serous from a pseudomucinous cyst are: The epithelial cells are high, cylindrical and ciliated. They possess large nuclei, centrally located, which fill out the greater part of the cell. The internal pressure is not so great in this variety as in the preceding.

The secretion is thin, not stringy and mucous, rich in albumin and of a yellow color, which may be changed by hemorrhages. The serous cystadenomata have a pronounced tendency to form papillæ. On section smaller or larger wart-like growths are found on the inner surface of the different cavities. They are composed of a mass of epithelial cells. These warts have a great tendency to proliferate and soon fill out the entire cyst cavity. They perforate the walls of the tumor and grow out over its surface. In some cases, the papillæ may primarily grow on the surface of the ovary, where they are termed surface papillomata.

The surface papillomata are spread by the peristalsis of the intestines through the entire abdominal cavity. They implant themselves on the parietal and visceral peritoneum, without having an anatomically malignant structure. The warts continue to secrete large amounts of a serous fluid leading to an extreme ascites.

The ascites is characteristic of the serous papillary

cystadenomata. These tumors, though anatomically benign are clinically only conditionally benign on account of their tendency to rapid dissemination and excessive proliferation. Serous cystadenomata are usually bilateral. At first one ovary becomes diseased and shortly afterwards the other, so that two different stages of the disease are met with coincidently.

3. Carcinoma Ovarii.

Ovarian carcinomata are relatively frequent. We distinguish three forms: (a) primary ovarian carcinomata, (b) carcinomatously degenerated serous and pseudo-mucinous cystadenomata and embryomata and (c) metastatic ovarian carcinomata derived usually from primary cancers of the digestive tract.

(a) *Primary ovarian carcinomata* develop from the germinal cells which deeply penetrate the ovarian tissue. The ovary usually retains its form. The tumor masses are nodular, their surface smooth. On section the ovary is seen to be firm, the connective tissue network may be recognized in whose meshes the cancerous tissues lie. If the interstitial tissue predominates it is designated as a hard scirrhus, if the cancerous tissue predominates, a soft medullary carcinoma.

Cysts may form in the tumor masses on account of necrosis or hemorrhages occurring in them. The cancer may involve the connective tissue capsule of the ovary and invade the peritoneum, causing a general peritoneal cancer with hemorrhagic ascites. Metastases may occur in the retro-peritoneal lymph nodes and also by way of the blood and lymph vessels in distant organs. Cachexia rapidly causes death of the victim.

On *microscopical examination* the carcinoma is seen to be either alveolar when nests of cancer cells lie between the connective tissue meshwork, or glandular when the cancer forms glandular tubules lined with stratified,

polymorphous epithelial cells. Ovarian cancers are, as a rule, bilateral.

(b) *Carcinomatous degeneration of ovarian cysts and embryomata* occurs but rarely in the pseudo-mucinous cysts and cystic embryomata, and more frequently in the serous cysts and solid embryomata.

In the pseudo-mucinous cystadenoma, portions of medullary consistency, greyish-white in color, having the form of an adeno-carcinoma, are found. The tumor, otherwise, retains the characteristics of a pseudo-mucinous cystoma. Gradually the entire tumor degenerates. The course is the same as that of primary ovarian cancer.

The serous papillary cystadenomata frequently undergo carcinomatous degeneration. The papillæ resemble macroscopically the benign form. On section, medullary masses penetrate the connective tissue. Microscopically the papillæ are constructed not of simple ciliated epithelium, but of stratified, polymorphous epithelium, which forms columns and medullary duct-like masses penetrating into the deeper portions of the tumor stroma. Rapid growth, dissemination into the peritoneum, with ascites and metastases, are characteristic.

In cystic embryomata, the epidermal portion may degenerate to form squamous epithelial-cell cancer with the same end results as other ovarian cancers.

(c) *Metastastic ovarian cancers.* The primary cancers are mostly located in the gastro-intestinal tract, more rarely in the uterus, the mammæ and so forth. The first symptoms often appear from the ovarian metastases and are enlargement of the abdomen and ascites. Even post mortem, it is difficult to locate the primary cancer, which may be a small carcinomatous ulcer of the bowel or a mammary nodule. The ovarian metastasis has the same microscopical characteristics as the primary cancer and usually occurs bilaterally.

II. Connective Tissue Ovarian Tumors.

Ovarian desmoids develop from the connective and muscle tissue of the ovary. They are less frequent than epithelial cell ovarian tumors.

The different forms are fibromata and sarcomata. Enchondromata, endo- and peritheliomata also occur, but very infrequently, and are highly malignant.

Ovarian fibromata are relatively frequent. They originate from a proliferation of the ovarian connective tissue and grow to a considerable size, two to three times the size of a human head. They are of a solid and either a hard or soft consistency, depending on the degree of density of the connective tissue, and have a smooth surface. Microscopically, irregular bundles of spindle-shaped cells, with more or less fibrillary tissue, are seen. Edematous infiltration, apoplectic foci, and necrotic softened areas occur as a result of circulatory disturbances or axial rotation.

Pedunculated fibromata the size of a cherry or hazelnut, and attached to the ovary by a narrow pedicle, may be accidentally found. They are not of any importance and originate from cicatricial tissue, caused by the contraction of corpora lutea. They hardly ever grow and are symptomless.

Fibromyomata are relatively rare. They originate in the vessel walls, are usually unilateral and contain smooth muscle tissue held together by connective tissue.

Sarcomata of the ovary are also infrequent. They are either spindle or round celled, grow rapidly to a considerable size, are very malignant, early cause metastases by way of the blood stream, are almost always found in early life and are usually bilateral.

Other but rare varieties of connective tissue ovarian tumors are enchondromata, chondrosarcomata, osteosar-

comata, endotheliomata, and peritheliomata. They are all extremely malignant.

III. Embryonal Tumors of the Ovary.

We recognize two varieties, the cystic embryomata, formerly termed dermoids, and the solid embryomata, formerly known as teratomata.

Ovigenous ovarian tumors contain tissues of all the germinal layers, ectoderm, mesoderm and endoderm. The ectodermal tissues usually predominate. The theories of their origin are that the tumors result either from a proliferation of the primary follicles which by partho-genesis form the different embryonal tissues (partho-genesis, however, never occurs in mammalia—Marchand); or (2) from inclusions of fecundated polar globules or impregnated blastomeres (Bonnet). The detached fecundated blastomeres would be taken up by the Wolffian body or mesoderm. The earlier the inclusion occurs and the less advanced the formation of the germinal layers has been, so much more varied tissue forms will arise. The later the process of detachment of the blastomeres takes place, i. e., the further the differentiation of the germinal layers has advanced, the fewer varieties of tissue can later be produced. The predominance of these tumors in the ovaries, though they may occur in other parts of the body, is explained by the fact that the mezonephros out of which the ovary forms reaches through almost the entire length of the fetus in its earlier stage, thus the best opportunity for the inclusion is offered in this structure. Accordingly all embryomata are congenital. They begin to grow under the influence of the regular recurring hyperemia of the pelvic organs during menstruation. These tumors have been found in young girls.

Cystic embryomata occur more frequently than the solid. They form round, movable tumors with smooth

surfaces and vary in size from a fist to a child's head. As a rule, one solitary cyst exists in whose walls are embedded the embryonal structures. The cysts are sometimes multilocular, when they are pseudo-mucinous. All the embryonal layers may be present. The ovigenous tissue consists of a small mass which projects into the lumen of the cyst. The mass is covered with epidermis. The wall of the cyst is formed of simple cylindrical (pseudo-mucinous) epithelium.

The epidermis in some places is covered with (usually) blond hair, the latter lying like coils in the cavity. The cyst is filled with sebaceous matter secreted by the epidermis, which remains oily at the temperature of the body and becomes a fatty, solid, white-grey substance on cooling. Teeth are rarely seen. Islands of cartilage and bone, even well-developed bones, as the maxilla and flat skull bones, may be present. All transitional stages of development, from rudimentary to almost completely developed organs, are met with. The ectoblast is represented by epidermis, teeth, nerve and brain tissue, the mesoblast by connective, adipose and muscle tissue, cartilage and bone, and the endoblast by intestinal tubes lined with cylindrical epithelium and often surrounded by circular muscle fibres. The tissues always show regularity in development. The growth of cystic embryomata is very slow. They are mostly unilateral, rarely bilateral and have an absolutely benign character, but may undergo degeneration into squamous celled epithelial cancers.

Solid Embryomata. (*Teratomata.*) These neoplasms are of much rarer occurrence than the cystic. Genetically they resemble the cystic embryomata; morphologically they differ.

Solid embryomata grow very rapidly and may attain a large size. They are composed of an irregular conglomeration of the most varied derivatives of all three germinal layers. The ecto- and mesoblasts predominate, espe-

cially bony tissue. Teratomata have a smooth surface with eminences and grooves. On section epidermal tissue, represented by hair and pigments, and mesoblastic tissue by bones and cartilage, are found. Microscopically the central nervous system besides the epidermis is oftenest represented. The solid embryomata are only conditionally malignant.

The Pedicle Formation of Ovarian Tumors.

The pedicle of ovarian tumors which grow towards the peritoneal cavity is formed from the ovarian ligaments, i. e., towards the pelvic bone by the suspensory ligament, towards the uterus by the utero-ovarian ligament, and towards the broad ligament by the hilus ovarii. The tube is located either beside or underneath the ovarian tumor without participating in its growth, or when its mesosalpinx is short, it may run on the surface of the tumor and become stretched to a very considerable length.

The pedicle is usually ligamentous and about five to six centimeters broad. It may stretch, when it decreases to the thickness of a finger. Ovarian tumors also may develop towards the hilus between the folds of the meso-ovarium—an intra-ligamentous development. This frequently occurs with the serous cystadenomata. It leads to a marked change in the position of the other pelvic organs and displaces the uterus and bladder out of the small pelvis and seriously compresses the vagina, rectum and ureter. With abdominal development the tumors at the beginning lie to one side of the pelvis in the excavatio recto-uterina. If their size fills out the space in the small pelvis they ascend into the large pelvis, where they locate always in the middle and anterior portion, so that the intestines lie posterior to them. If the tumor continues to grow, it gradually occupies the entire abdominal cavity, presses on the stomach and liver, elevates the diaphragm, compresses the lungs, and displaces the heart.

The presence of such a large foreign body causes changes in the peritoneum. Pressure from the tumor results in a destruction of its surface epithelium and the endothelium. An exudation follows causing an agglutination, and finally connective tissue adhesions between peritoneum and tumor wall. Such adhesions may also result from infections derived from the bowels or the tube. The peritoneum and tubal walls sometimes suffer a marked increase in thickness on account of the chronic irritation caused by pressure. This considerably lowers the resistance of the peritoneum to the dangers of an operation (peritonitis).

Torsion of Pedicle. Axial Rotation. As long as the tumor possesses a certain mobility, it may turn around its axis (axial rotation) during changes of position, or from exertion of its bearer, and the pedicle suffers a torsion. This results in a compression of the blood vessels. The veins are the first to become obstructed, with the consequence of a venous stasis in the tumor, which causes the latter to rapidly enlarge to twice its size or more. Finally, if the torsion continues, the arterial blood current is also interrupted and the tumor becomes entirely cut off from the blood circulation, and a necrobiosis of the mass results. Thereby the interior of solid new growths softens and breaks down. Thus large cysts may occur in solid tumors. An ovarian tumor with a twisted pedicle is darkish-blue. The pedicle and the tube also become enlarged. A necrobiotic neoplasm causes a circumscribed fibrinous peritonitis which finally surrounds the entire tumor with adhesions. A new blood supply is established through these adhesions assuring the continued existence of the tumor. At other times the tumor may become entirely detached from its pedicle, become surrounded by peritoneal adhesions and remain in the peritoneum as a foreign body, and if its proliferative energy has become

extinct, it undergoes mummification or calcareous degeneration.

During the circulatory disturbances of ovarian tumors, and even without them, edema, hemorrhages, fatty and myxomatous degeneration, necrosis, or calcification may take place. Calcareous deposits in the tumor are designated as psammo-bodies (psammo-cystoma, psammo-carcinoma).

Ovarian tumors may become infected, the infectious material being derived from puerperal fever, pneumonia, typhoid fever, tuberculosis and also from adherent intestines. Infections usually terminate in suppuration and putrefaction of the tumor.

Parovarian Cysts.

Parovarian cysts are retention cysts and not new growths. They are benign and result from an increased secretion of the parovarian ducts, which causes a distension of the different glandular canals and this leads to the formation of a unilocular cyst.

These cysts are extra-peritoneal. The wall consists of peritoneum, a layer of connective tissue with a few smooth muscle fibres, and an inner lining of simple cylindrical epithelium. Warty excrescences may be formed.

Parovarian cysts are usually small. They are not intimately connected with the uterus or broad ligament. Rarely they may become pedunculated when axial rotation may cause a torsion of the pedicle leading to all the complications described above.

Clinical History of Ovarian Tumors.

Symptoms. Ovarian tumors develop during the child-bearing period, but childhood is not exempt from them. They may grow to a large size without causing any disturbances.

The earliest noticeable symptom is a feeling of *restriction of space* in the pelvis, which is characterized by a

sensation of fullness and pressure in the abdomen and an interference with the function of the bowels and bladder. With an intra-pelvic position the tumors are seldom visible; with abdominal position, usually. As the tumors grow, aggravation of the symptoms of restriction of space and a tenseness of the abdominal walls result. An edema of the vulva and of the dependant portions of the abdominal wall, and finally of the legs, develops from an obstruction by pressure of the return circulation.

The umbilicus becomes obliterated, and an umbilical hernia may form. At times the breasts enlarge and form colostrum. In advanced stages a cachexia occurs which is favored by anorexia, discomfort, pain, inability to lie comfortably and disturbed sleep. The suffering expression of the countenance has been justly termed *facies ovarica*. The cachexia is characterized by an atrophy of the muscular system, loss of strength and a dry, harsh skin. The patient finally succumbs to dyspnea and cardiac weakness.

Menstruation may remain normal. With complete bilateral degeneration we find amenorrhea. Absence of menses also results from advanced debility.

Ovarian tumors do not preclude pregnancy.

The course of the disease is usually slow and progressive. It becomes rapid if malignancy be present.

The diagnosis of ovarian tumors differs according to their location and size. We will, therefore, consider the diagnosis in three sections: (1) Small ovarian tumors located in the true pelvis. (2) Medium-sized ovarian tumors reaching up to the umbilicus and located in the abdomen. (3) Large tumors filling the abdominal cavity.

1. Bimanual examination reveals the tumor in the posterior cul-de-sac. It is round, smooth, tense, insensible to pressure and usually freely movable. According to its location and size the uterus becomes displaced. The tumor displaces the uterus especially if it is adherent or intra-

ligamentous. The latter are always immovable. If the tumor is incarcerated in the true pelvis an edema of the vagina and the pelvic floor results.

A differential diagnosis must be made between inflammatory ovarian disease, extra-uterine pregnancy, hematoma, hematocele, para- and perimetritis and myoma uteri. (See table 2.)

2. Medium-sized ovarian tumors. The contour of the abdomen is semi-spherical. It is enlarged. The tumor lies immediately beneath the abdominal wall and anterior to the intestines. Bimanually the tumor is found to be connected with the pelvic organs. Dullness is present over the tumor and tympany laterally to and above the mass. The uterus is displaced downwards either in ante- or retro-position. Fluctuation may be elicited. The pedicle may be located by displacing the uterus downward, when the pedicle is felt as a tense band.

Differential diagnosis should be made between 6th to 7th month pregnancy, myomata and tumors of other abdominal organs. (See table 3.)

3. Large ovarian tumors which fill out the entire abdomen. The form of the abdomen is dome-like and does not change with a change in the position of the patient. The abdominal walls are movable over the tumor surface, except when peritoneal adhesions exist. The percussion sound is flat, fluctuation is present and auscultation is negative.

The differential diagnosis between ascites, pregnancy, other abdominal tumors (liver, pancreas, spleen, which move with respiratory movements) and phantom tumors is to be made. (See table 4.)

Differential diagnosis between the different varieties of ovarian tumors is many times impracticable, usually impossible and always unnecessary as the diagnosis of ovarian tumor means its removal. Carcinomatous degeneration must be suspected when sudden acceleration

in the growth of the tumor occurs, with increased pain, ascites and anasarca, cachexia and the rapid loss of weight. Infection of ovarian tumors is ushered in with chills, fever, hyper-leucocytosis and increased pulsation of the pelvic blood vessels. Aspiration or paracentesis may clear up the diagnosis, which procedure, however, is dangerous and should be immediately followed by drainage, if an abscess is present.

Torsion of the pedicle is indicated by sudden, stormy symptoms, as severe pain, tympanites, chills, fever, vomiting, shock, and enlargement of the tumor.

Rupture of an ovarian cyst is dangerous. The cellular elements become engrafted on the peritoneum and form secondary growths. Otherwise the fluid becomes absorbed, the cyst wall closes and the cavity refills.

Pregnancy may occur in the presence of an ovarian tumor. The former causes the latter to grow at an accelerated rate during its existence. If the tumor lies in the small pelvis it does not hinder the development of the uterus, but obstructs the passage of the child during labor. If the tumor rises with the uterus into the abdominal cavity, it may impede the development of the uterus by pressure, causing premature expulsion of the fetus.

The prognosis must be guarded under all circumstances. In solid embryomata, serous cysts and malignant growths the prognosis is serious. The course of pseudo-mucinous cysts is usually favorable, however, it may be, in exceptional cases, as bad as the foregoing.

The treatment is surgical. In young people, in the absence of malignancy, the fellow ovary may be saved if it is found normal. If carcinoma or sarcoma exist an extended total radical extirpation should be performed. Other tumors must be removed abdominally. The pedicle is clamped and severed, the growth removed, the stump ligated and covered with peritoneum or fat.

Some surgeons aspirate the contents of the cyst through a large trocar to obviate an extensive incision. The contents of the cyst is usually spilled into the peritoneal cavity and a danger of the formation of secondary growths in the abdominal cavity is thereby possible. For this reason a large incision is preferable, if necessary, reaching from the symphysis pubis to the xiphoid cartilage, to remove the growth with its contents *in toto*. In small cystic degenerations, pseudo-mucinous, dermoid, and parovarian cysts of the diseased ovary the opposite ovary should be saved. In serous cysts the other ovary should be removed. We should not subject the patient to the added risk of another tumor-formation in the remaining ovary.

CHAPTER XI.

DISEASES OF THE PELVIC CONNECTIVE TISSUE.

Parauterine Extra-peritoneal Hematoma.

Hematoma extraperitoneale parauterinum. Pelvic hematoma.

Etiology. Injuries during labor and gynecological operations are the most frequent causative factors of pelvic hematomata. Non-puerperal hematoma occurs once in about one thousand gynecological cases. Predisposing causes are pre-menstrual hyperemia, varicosity of the pelvic blood vessels during pregnancy and labor, scar formation in the pelvic connective tissue and cardiac diseases. Other direct causes are concussions of the abdomen, blows, suddenly increased intra-abdominal pressure, violent and perverted coitus and masturbation with foreign objects. Finally extra-uterine pregnancies may rupture between the folds of the broad ligament. Gynecological operations cause hematoma by trauma and imperfect or careless ligation of bleeding vessels.

Pathology. If a rupture of a blood vessel occurs beneath the pelvic diaphragm a para-vaginal hematoma forms, if above the diaphragm an intra-ligamentous hematoma. The latter are the most important. The blood advances in the direction of least resistance and usually remains extra-peritoneal. It may invade the mesosigmoid, follow the ureter and ovarian vessels to the kidney, or extend downwards towards Poupart's liga-

ment or anteriorly towards the bladder. A hematoma is usually unilateral. It may also be bilateral, or two hematomata may occur, one on each side.

In the majority of cases of hematomata an absorption of the blood takes place. Infection may occur, leading to suppuration. The abscess may rupture into the hollow organs or outwardly.

The symptoms consist of *pain* of varying intensity and a feeling of pressure in the pelvis, with interference in the functions of the bladder and rectum. *Anemia* is present to a slight extent, its degree depending on the amount of blood lost. *Shock*, vomiting and so forth also occur. *Menstruation* appears prematurely or may be delayed, the amount of menstrual fluid may be increased or diminished.

Signs. A mass is felt which is sensitive to pressure, and which varies in size from a hen's egg to a man's head. It is located laterally in the pelvis and may reach up to the umbilicus. The uterus is displaced towards the opposite side, either forward or backward, and it remains somewhat movable. If the hematoma is bilateral, the uterus is located medially and appears to be walled in, so that it is hardly possible to outline the organ.

The hematoma is not sharply-defined. The adnexa of the opposite side are distinctly palpable, if the hematoma is unilateral. The rectum becomes displaced. Loops of intestines usually found present in the recto-uterine pouch are displaced upwards. The sacro-uterine ligaments are forced together. The blood mass is soft and doughy and liquefies gradually.

Course. The symptoms and signs decrease and finally subside. The anemia improves and with it the general constitutional symptoms. The tumor disappears by organization or absorption.

If infection results, rigors with pyrexia and symptoms of sepsis appear. The abscess ruptures sooner or later

and an ill-smelling, brownish substance, mixed with blood clots, is discharged.

The diagnosis is made from the history of trauma, the symptoms and the bimanual findings.

Hematoma should be differentiated from an hematocele, a parametritis, a perimetritis, intra-ligamentary myoma uteri and adherent ovarian tumors. (See table 7.)

The prognosis should be guarded, though it usually is favorable. Absorption is completed within six to eight weeks.

The treatment consists of absolute rest in bed, elevation of the head of the bed, the application of an ice-bag to the abdomen and the internal or subcutaneous administration of narcotics to relieve severe pain. Within three or four weeks absorption may be promoted by iodin vaginal douches, the insertion of ichthyol tampons and the use of Bier's hyperemic treatment.

An uninterrupted, continued growth of the mass, with an increasing anemia as determined by a hemanalysis and indicating a continued bleeding, necessitates opening of the hematoma and ligation of the bleeding points to arrest the hemorrhage. Lowly-situated tumors may be attacked through the vagina, large, highly-situated ones through the abdominal wall. The latter, however, is the preferable route.

If suppuration occurs, drainage, either through the vagina or the anterior abdominal wall, depending on the location of the mass, is indicated. The vaginal route is preferable.

Parametritis.

Etiology. Parametritis is always due to an infection, which in the majority of cases ascends from the genitalia, but may also descend from the bowels or bladder or be hematogenous. It is observed in the majority of cases in connection with labor and the puerperium.

Table No. 7—Differential Diagnosis of Pelvic Hematoma. See also Table 8.

	Hematoma.	Hematocele.	Parametritis.	Intraligamentous myoma uteri.	Adherent ovarian tumor.
History.	Sudden onset after labor, trauma or operation.	Same but especially ruptured tubal pregnancy.	Same plus history of infection, also extra-genital focus of infection.	Insidious onset plus menstrual disturbances, especially menorrhagia.	Insidious onset and history of pelvic peritonitis. Menses normal.
Symptoms.	Pain-pressure, mild degree of anemia.	Pain; symptoms of intra-abdominal hemorrhage. Temp. decreases and pulse-rate increases.	Sepsis; pyrexia, rapid pulse, severe pain.	Pressure symptoms plus menstrual disturbances, especially menorrhagia.	Referable to peritonitis: pain, sensitiveness.
Signs. a. Location.	Laterally to uterus. Opposite adnexa are usually palpable. Uterus fixed to the exudate.	Blood mass located centrally in recto-uterine pouch and posterior to uterus. Latter pushed forward and limited in movability.	Lateral to uterus and fixed to it. Uterus immovable. Opposite adnexa palpable.	Lateral to uterus. Uterus connected to it. Therefore limited movability. Adnexa palpable.	Lateral and posterior to uterus. Uterus limited in motion; opposite adnexa palpable.
b. Tumor mass.	Hard, firm, somewhat sensitive. Later fluctuating. Ill-defined. Gradually absorbed and disappears.	Soft, liquid, markedly sensitive. Gradually absorbed and disappears.	Same as hematoma but very sensitive, either undergoes resolution or suppurates.	Sharply defined nodular, hard. Not sensitive to pressure. Immovable. Continues to grow.	Hard or semi-solid. Well-defined, immovable, somewhat sensitive. Continues to grow.
c. Sacro-uterine ligaments.	Forced together. Mass surrounds the rectum.	Forced apart. Mass compresses rectum.	Forced together. Mass surrounds rectum.	Forced together. Mass compresses rectum.	Forced together or apart. Mass compresses rectum.
Blood count.	Slight degree of anemia.	Severe anemia.	Hyper-leucocytosis and increase in neutrophiles.	Negative. May have anemia with profuse menorrhagias.	Negative.

Pathology. The exudation forms, with predilection, in the cardinal ligament from whence it advances towards the posterior and lateral pelvic cellular tissue. The exudate is intimately connected with the cervix, and both present a rigid, inflexible mass.

The exudate may develop, but rarely, in the upper portion of the broad ligament by a continuation of an infection from the corpus uteri. Thence it may spread towards the iliac fossa, leaving free the lower part of the pelvic connective tissue.

The exudate may also extend along the sacro-uterine ligaments into the septum recto-vaginale, or to the pre-cervical or para-cervical connective tissue, or the pre-peritoneal cavity of Retzius, or to the subserosum of the anterior abdominal wall.

The infective organisms are chiefly the streptococci pyogenes, but staphylococci, bacilli coli and rarely gonococci, actinomycoses and at times bacilli tuberculosis may be the invading organisms.

The following *varieties* may be noted: (1) Acute: (a) acute septic puerperal parametritis, (b) acute septic traumatic parametritis, with which we class the gonorrheal, and (c) acute extragenital parametritis originating from infectious foci external to the genital organs, i. e., descending and hematogenous infections. (2) Chronic: (a) chronic puerperal parametritis, (b) chronic traumatic parametritis, (c) chronic extragenital or descending parametritis and (d) chronic atrophic parametritis.

Acute septic puerperal parametritis is the most frequent and most important form of infection of the pelvic connective tissue.

The symptoms are ushered in by rigors, followed by an elevation of temperature and increase of pulse-rate. Pulse and temperature run parallel to each other. They gradually decline after four to seven days, when resolu-

tion begins, or they drop abruptly to normal after the escape of the pus, if an abscess has formed.

Pain may precede every other symptom. It is felt in the bladder, bowels and peritoneum. The patient immediately becomes profoundly ill. The disease usually terminates, after a few days of stormy symptoms, with a complete resolution. Exceptionally an abscess may form. The abscess may rupture into the vagina or through the anterior abdominal wall, depending on the location of the exudate. It may also open into the abdominal cavity, causing a rapidly progressing, fatal peritonitis. This accident, however, is infrequent. Parametritic exudates may cause a thrombosis of the femoral vein, terminating in a phlegmasia alba dolens.

Signs. On bimanual examination a marked sensitiveness is felt over a doughy mass extending laterally from the cervix towards either side. The exudate may fill out the entire posterior pelvic cavity. The vaginal vault of the affected side bulges. The mass becomes firm and hard as a board, and is not sharply outlined. The rectum is displaced laterally. The bladder becomes compressed. The sacro-uterine ligaments are forced together.

Absorption occurs from without inward, i. e., the portion next the uterus is the last absorbed. The adnexa become palpable with a progressing resolution of the exudate.

Suppuration is indicated by continued pain, temperature, increased pulse-rate and persistent leucocytosis.

Differential diagnosis. Acute septic puerperal parametritis is differentiated from the other forms by the history of the case. From salpingitis and oophoritis, by the fact that in parametritis the sacro-uterine ligaments are pressed together, while in intra-peritoneal pathological conditions they are forced apart and a bulging is observed in the posterior vaginal vault.

Differentiation from hematocele and extra-uterine

pregnancy is the same as for hematoma. Tumors of the uterus and ovaries are never accompanied by fever, and seldom by pain, excepting pressure-pains. Exudates are absorbed, tumors never. (See table 7.)

Chronic septic puerperal parametritis follows the acute form if resolution is retarded or does not take place.

The symptoms are dysuria, constipation, dysmenorrhea and sterility. If pregnancy occurs, it is usually terminated by abortion, with septic fever following.

The *prognosis of the acute form* is grave, but not absolutely unfavorable. It depends upon the virulence and number of the causative germs, the condition of the pelvic connective tissue and the resistance of the patient. It is absolutely bad if a general sepsis or an invasion of the peritoneum occurs. The *prognosis of the chronic form* is bad as regards the re-establishment of the normal functions of the pelvic organs.

Treatment. Prophylaxis. Strictest asepsis must be observed during labor and the puerperium, and repeated, unnecessary examinations, and instrumental deliveries, should be avoided.

The treatment of the acute form consists of absolute rest in bed, elevation of the head of the bed, ice bags to the abdomen, sponging for temperature, antipyrin to reduce fever, gastric lavage for nausea and vomiting and narcotics for pain.

Rectal feeding should be instituted if symptoms of peritoneal irritation exist. Otherwise give nourishing broths, meat juices, buttermilk and kumiss. If the patient is profoundly ill, or septic, normal saline solutions should be given per rectum or subcutaneously, with the addition of one dram of glucose to each quart of solution.

After the fever has been absent for eight days, the patient may leave the bed. We should now order iodin vaginal douches at 120 degrees F., also full baths, Sitz

baths, salt baths and, after another four to six weeks, hot-air baths.

As soon as suppuration takes place, drainage is indicated. Abscesses pointing towards the vagina are to be opened by a posterior colpotomy, abscesses located above the brim of the pelvis should be evacuated through a low incision in the anterior abdominal wall, if possible over the middle of the mass.

(b) **Acute septic traumatic parametritis** is caused by infections following trauma, as surgical operations, gynecological examinations, contusions from blows, coitus and cancers or necrotic tumors.

The exudation surrounds, at first, the area of the trauma, the injured tissue becoming necrotic. It may spread and involve the entire parametrium.

Symptoms develop either rapidly or slowly. They are ushered in with rigors and pains, followed by pyrexia, acceleration of the pulse and corresponding prostration. Involvement of the peritoneum is indicated by nausea, vomiting, tympanites, painful urination, obstinate constipation, and pseudo-ileus. The disease may terminate by resolution or suppuration.

Signs. The tumefaction at the seat of the wound and the adjacent parts is very sensitive. The mass is hard, edematous and spreads rapidly to the pelvic brim and iliac fossa. The rectum is compressed, the bladder displaced upward, and the uterus and adnexa are hardly palpable. Abatement of the fever and pain indicates resolution; an increase of fever and pain, however, points toward suppuration. Resolution is complete with a thorough recovery, or incomplete when indurated peritoneal bands and scars remain, causing continued pains, interference with the function of the pelvic organs, such as dysmenorrhea and rectal and vesical tenesmus.

The cicatrices and adhesions may displace the uterus, interfere with its circulation and cause sterility. Gonor-

rheal parametritis may occur, but it is very rare. Gonococci must then be demonstrated by the microscope.

The prognosis should be guarded, depending on a complete or incomplete resolution or suppuration. With suppuration, post-operative hernias at the seat of the incision and drainage are frequent. The results of plastic operations, if followed by parametritis, are negatived.

The treatment is the same as in the puerperal form. Operation wounds should be reopened if fluctuation is imminent. The gonorrheal form should be treated in the same way. If an abscess forms, open it and drain.

(c) **Acute extragenital parametritis** is the result of a descending and contiguous infection from the bladder, bowels, bones, adnexal organs. The infection invades the parametrium either by continuity or through the blood or lymph vessels. Cystitis, proctitis, typhlitis, perityphlitis, appendicitis, sigmoiditis, pyonephrosis, nephritic abscesses, empyema of the gall-bladder, osteo-myelitis, periostitis and coxitis of the pelvic bones may cause a parametritis.

The clinical picture is preceded by that of the primary disease. Prognosis and treatment depend on that of the primary disease, plus the parametritis.

Chronic Atrophic Parametritis.

This is a senile atrophic process occurring shortly before or during the climacterium. It is the result of a pelvic connective tissue inflammation without any known cause. Atrophic parametritis may result from general disturbances of nutrition (anemia, tuberculosis, infantilism) repeated and frequent pregnancies, difficult labors complicated by profuse hemorrhages, improper nourishment of the ganglia and nerves of the pelvic connective tissue.

The *symptoms* are pain and neuroses due to a neurasthenia of the patient. The nerve manifestations are

either a sympathetic, spinal, or cerebral hysteria, the first causing celiac neuralgia, the second, sciatica, backache, arthropathy and interference with sensibility and mobility, and the third, trigeminal neuralgia, headache, disturbances of vision, copiopsia (eye-strain), hysteria and vasomotor disturbances.

The signs consist of an emaciation of the entire pelvis, atrophy of the genital organs and premature senility.

The *prognosis* is doubtful, depending on the possibility of the restoration of the general nutrition of the body.

The *treatment* is symptomatic. We must abstain from all local gynecological treatment, except if adnexal disease is present which requires removal. For profuse leucorrhea and hemorrhages use curettage and, finally, hysterectomy.

In the absence of any other pathological condition of the pelvic organs gynecological treatment is contra-indicated and ill-advised.

CHAPTER XII.

DISEASES OF THE PELVIC PERITONEUM.

Pelvic Hematocele.

Definition. An effusion of blood into the pelvic peritoneal cavity.

Etiology. This is a disease of women in the prime of life and may be caused by (1) venous stasis or passive congestion occurring, for instance, during typhus, cholera, poisoning (phosphorus), burns, purpura, Bright's disease and so forth; (2) rupture of an ovarian follicle or a reverse flow of blood through the tubes; (3) injuries to the genitalia and gynecological operations; (4) the most frequent cause, however, is a rupture or abortion of an extra-uterine pregnancy.

Pathology. The effusion of blood collects in the recto-uterine pouch. If the pouch, however, is occupied by a retro-flexed uterus or a tumor, or obliterated by pelvic adhesions, the blood collects in the para-uterine or vesico-uterine pouches. The absorptive power of the normal healthy peritoneum rapidly removes the blood, while in an inflamed peritoneum a blood-tumor is formed. The coagulated blood then undergoes organization and absorption.

Under certain circumstances the outer layer of the blood clot is formed by hard, tough fibrin, a capsule, which is of an almost whitish gloss, while the central parts become liquefied. Such a blood tumor is usually the cause of adhesions which have a tendency to persist after-

wards. Infection may occur in a hematocele, leading to an abscess formation. The patient may then succumb to sepsis or the abscess may rupture, usually through the vagina, bladder, rectum or anterior abdominal wall. It rarely ruptures into the free peritoneal cavity.

Subjective symptoms. These depend on the causes. If the hematocele is due to a passive hyperemia, the symptoms are slight, if due to trauma or injury, they consist of shock, pain, peritoneal irritation, followed by anemia, feeling of weight and pressure (limitation of space) and an interference with the functions of the bladder and bowels.

Infection is ushered in by an initial chill, followed by fever, rapid pulse, pain, and a leucocytosis characterized by a percentual increase of neutrophiles.

Objective signs. As long as the blood is liquid the mass cannot be palpated. The uterus is displaced, the excavatio recto-uterina widened. Marked sensitiveness and a dull percussion note may be elicited over the hypogastric region.

If the blood coagulates, then the posterior vaginal vault bulges into the lumen of the vagina. The blood tumor is felt as a hard, doughy mass, in which are contained the adnexa so that they cannot be palpated. The uterus is felt anteriorly to the mass, is immovable and firmly pressed against the bladder and symphysis pubis. The blood mass is sensitive in the beginning. The sensitiveness gradually disappears with progressive organization. It reappears if an infection ensues, which fact is characterized by a hyper-leucocytosis, hyper-pyrexia and sepsis.

Interference with the bladder and rectum gradually disappears as absorption progresses. If absorption is delayed, chronic invalidism occurs, characterized by loss of strength, with profuse and painful menstruation and pain in the pelvis.

The subjective and objective signs of hematocele following tubal abortion or rupture are described in the chapter on diseases of the uterine tubes.

The *differential diagnosis* of pelvic hematocele considers pelvic peritonitis, pelvic hematoma, parametritis, retroflexed uteri, tumors other than those from genital organs, as rectal cancer, metastasis in pelvis, and, finally, displaced abdominal organs, especially kidneys and spleen. (See table 8.)

The *prognosis* depends on the cause, the amount of blood lost, and the possibility of absorption, organization and the occurrence of suppuration.

The hemorrhage may prove immediately fatal. Encapsulation of the blood clots and delayed organization lead to chronic invalidism. Future pregnancies may occur after recovery from hematocele.

Treatment. The object of treatment at the time of the occurrence of the hematocele consists in the arrest of the hemorrhage through a laparotomy incision. If the patient is seen after coagulation has occurred, rest in bed, ice to the abdomen and rectal feeding are the methods of choice. Should liquefaction occur, or the pressure of the mass cause unbearable suffering and organization does not follow, evacuation of the hematocele is then indicated. This is performed through the posterior vaginal fornix.

Continued ill-health and loss of earning capacity caused by peritoneal bands and arrested organization demand surgical interference. Suppuration indicates drainage. Hematocele due to extra-uterine pregnancy requires the same treatment.

Pelvic Peritonitis. Perimetritis.

Etiology. This is the most frequent of the diseases of the female pelvic organs and is due either to genital or extra-genital causes. From the acute it may become chronic. Perimetritis due to genital disease may occur

Table No. 8—Differential Diagnosis of Pelvic Hematocele and Perimetritis. See also Table 7.

	Pelvic hematocele.	Perimetritis.	Retroflexio uteri gravidi	Subserous myomata uteri.	Pelvic tumors not genital.
History.	Sudden onset after congestion, trauma and extrauterine pregnancy.	Sudden onset after labor, puerperium, therapeutic interference.	Amenorrhea and symptoms of pregnancy.	None. Perhaps gradually increasing menorrhagia.	Injury or cancer, and cachexia and age of patient.
Subjective symptoms.	Initial shock, peritoneal irritation. Symptoms of internal hemorrhage. Pressure symptoms.	Initial rigor, pyrexia, pain, nausea, vomiting, constipation, tympanites.	Ischuria, ischuria paradoxa, obstinate constipation.	Hemorrhages and pressure symptoms of long standing.	Pain of boring, intermittent character, gradual loss in weight and strength.
Objective symptoms.	Encapsulated mass slightly sensitive to pressure, posterior to uterus. Sacro-uterine ligaments forced apart. Organization slow.	Exudation. Signs of peritonitis sensitiveness, tympanites, uterus embedded in mass. Sacro-uterine ligaments forced apart. Rapid course.	Softening of uterus. Bluish discoloration. Enlarged breasts. Continuation of corpus with cervix. With correction of position continued growth.	Smooth, well-defined, hard mass attached to uterus. Uterus plainly palpable Adnexa palpable. Sacro-uterine ligaments forced apart. Continues to grow.	Uterus adherent to mass. Mass enlarges rapidly.
Blood examination.	Anemia, decrease in hemoglobin; oligocythemia.	Hyper-leucocytosis. Increase in neutrophiles.	Normal.	Normal.	Secondary anemia.
Abderhalden.	Negative, except if due to extrauterine pregnancy.	Negative, except if following labor or early puerperium.	Positive to placental tissue.	Positive to myoma tissue.	Positive with cancer tissue; negative with placental.

Other abdominal organs: Usually gradual in onset.

Subjective symptoms referable to displaced organ, as Dietl's crisis.

Objective symptoms: Outline of displaced organ and absence of the same in its normal location.

during intra-uterine life, it is then congenital. The predisposing causes are (1) hyperemia from ovulation, (2) the incidences of sexual life, (3) extra-uterine pregnancy, (4) tumors, (5) infections following labor, the puerperium, operations and gynecological treatment, (6) diseases of the bladder and bowels, and (7) salpingitis and endometritis.

Pathology. Acute form. An exudate forms which is either serous, fibrinous, hemorrhagic, purulent or ichorous (sapremic). The differences depend on the virulency and number of the infective germs and the resistance of the patient. The exudate becomes encapsulated, whence organization takes place with either a complete resolution or the exudate becomes absorbed, leaving connective tissue bands, adhesions, and thickening of the peritoneum. A great variety of pathological germs may be found in the exudate, even gonococci and colon bacilli, the latter oftener than the former.

The tubercle bacillus gains access to the pelvic peritoneum either by ascending from the tubes, or by the hematogenous route from the lungs or any other part, or the infection descends from the intestines and lymph glands. Tuberculous perimetritis may be of the dry form, characterized by an agglutination and adhesion of the organs by the tubercles, or it may be of the cheesy variety with abscess formation. The latter increases without limit, destroys the walls of the organs and often causes the formation of intestinal fistulæ. The cheesy form is usually found in children. Tuberculous peritonitis with serous, hemorrhagic, and purulent exudation is more frequently found in adults. Adhesions are infrequently found in the latter form. Healing takes place with the formation of scar tissue, fibrous adhesions, and calcareous depositions.

Chronic peritonitis follows the acute form. Resolution was incomplete and the pathogenic germs retained their

virulency. This results in especially heavy scar formation, the occurrence of fecal fistulæ with minute capillary openings, and a distortion of the entire pelvis. The pelvic organs become adherent to the intestines and rectum. The adhesions sometimes form broad bands of tissue, which overlie the organs like veils, the so-called pseudo-membranes. They hinder the absorption of the exudate and transudate.

Other forms of chronic pelvic peritonitis are pseudomyxoma peritonei after the rupture of a cystoma glandulare serosum, peritonitis carcinomatosa, and finally chorion-epithelioma peritonei following extra-uterine pregnancy.

Clinically we distinguish between (1) diffuse non-infectious perimetritis and (2) infectious perimetritis. The latter may be septic, sapremic, gonorrheic or tuberculous.

1. **Diffuse non-infectious peritonitis** is the result of mechanical or chemical irritation of the pelvic peritoneum by effusions of blood, myomatous tumors, and ovarian cysts. Sometimes the cause is undiscoverable.

Pathology. Diffuse non-infectious perimetritis is a localized disease process. The peritoneum is dark-blue in color, and is covered with fibrin that forms adhesions. The peritonitis remains confined to the fundus uteri, the anterior surface of the uterine body, the ovaries, and the para-rectal, para-uterine, and para-vesical fossæ. The adhesions are either firm and short, or thin, long and pseudo-membranous. The exudation is insignificant. It is a yellowish, serous fluid, rich in albumin, containing endothelial cells, but no bacteria. The exudation is enclosed in vesicles, *seroceles,* which are found on the uterus, ovaries, parietal and visceral peritoneum, omentum and even abdominal organs.

The symptoms are caused by the adhesions and vary according to their extent or firmness, resulting in an im-

mobilization or limitation of movability of the organs. Thus constipation and sterility are constant concomitant symptoms. There are present vesical and rectal tenesmus, dyspareunia and pain during physical exercise. The digestion is impaired, the patients gradually emaciate, complain of headache, are incapable of physical and mental exertion, and usually suffer from dysmenorrhea. Fever is absent.

The objective *signs* vary according to the location of the adhesions. The organs may be in a normal position and but little or not at all sensitive, or they may be distorted in form, adherent to each other and immovable. The peritoneum is thickened, causing an increase in the size of the organs. If an exudate is present, the seroceles may be felt as cysts.

The *diagnosis* is made from the history of the case, prolonged observation, palpatory findings, and apyrexia.

The *prognosis* should be invariably guarded. Adhesions are always serious.

Treatment includes an attempt at the promotion of absorption by iodin douches, tampons, massage and mechanical treatment, also mud-baths.

Continued ill-health and loss of working capacity indicate surgical interference, which consists in breaking up the adhesions and a removal of the products of inflammation. The denuded areas must be covered with peritoneum or fat tissue taken from the anterior abdominal wall.

Septic Peritonitis.

This follows labor, the puerperium, gynecological operations and treatment, and is caused by pus organisms (staphylococcus pyogenes and streptococci).

The *symptoms* are stormy, beginning with an initial chill, which is followed by fever, pain, tympanites, nausea, vomiting, ileus, dryness of the tongue and lips.

The pulse curve is analogous to the temperature curve. Cardiac weakness is early manifested.

Course. Septic peritonitis is usually fatal within one week. If improvement ensues, the virulency of the germ decreases, the exudation is discharged by rupture, either natural or artificial, the temperature subsides, the pulse slows down and improves in quality. Peristalsis is re-established. Convalescence and resolution are often interrupted by repeated acute exacerbations which may eventually prove fatal.

Sapremic peritonitis is an intoxication due to the absorption of bacterial putrefactive products.

The *symptoms* are irregular temperature and a continual small and frequent pulse. The incongruence of temperature and pulse is characteristic of sapremic conditions.

Dyspnea and rapid respirations, delirium and coma occur early. Ileus or exhausting diarrheas interchange. A stale, fetid odor from the mouth is conspicuous. The exudation varies in amount and becomes usually purulent. If free drainage cannot be secured, the patients usually succumb to a chronic sepsis.

Gonorrheal Perimetritis.

There may be a very stormy peritonitis, or a slow process. After a violent beginning, a complete resolution may occur. Extensive exudations seldom form. They become organized, leaving adhesions behind.

Restitutio ad integrum is not unusual. Such women may become pregnant and have normal labors and puerperii.

Tuberculous perimetritis has been fully considered under tuberculosis of the female genital organs.

The *diagnosis* of all the infectious forms of perimetritis is made from the history of the case together with

the symptoms and course. Chronic perimetritides are with difficulty diagnosed.

Signs. Adhesions which cause functional disturbances of organs, are often palpable. Exudations are chiefly found in the recto-uterine pouch. If acute, they are sensitive, if chronic, less so. The exudations are movable, of hard consistency and from the size of a hen's egg to that of a large ovarian tumor. They push down the posterior vaginal vault, displace the uterus forward, force the sacro-uterine ligaments widely apart and surround and compress the rectum. Ovaries and tubes are enclosed within the exudate. The latter decreases progressively in size, in contradistinction to tumors, which gradually increase in size.

Abscess formation results in softening of the exudation. It finally ruptures if left undisturbed. Irregular temperature and leucocytosis are suspicious signs of it.

Gonorrheal perimetritis is diagnosed from the history of the case, the admittance of such an infection and the microscopical examination of uterine and vaginal discharges.

Tuberculous peritonitis is characterized by a slow onset, amenorrhea, the existence of a primary focus elsewhere, afternoon temperature, loss of strength, age of patient, and, finally, diagnostic tuberculin injection.

The prognosis of acute septic and sapremic peritonitis is always grave. An early evacuation of the exudate improves it. Permanent recovery is doubtful on account of the adhesions which lead to functional and organic disturbances.

Gonorrheal perimetritis offers a bad prognosis although restitutio ad integrum often occurs.

The prognosis of tuberculous peritonitis depends on the extent of local and other processes.

Treatment of acute perimetritis. Exudation demands drainage. The after-treatment, after opening the ab-

scess, includes transfusions of 500 cc. of a solution of sodium chlorid 5.9, glucose 38.8, and water 1000. The transfusion may be repeated every four hours for three times, or the saline solution may be given as a continuous proctoclysis. Tuberculous perimetritis demands, in addition, proper antiphthisic measures, as tuberculin injections, internal administration of black drops and open air treatment. (Black drops are compounded as follows: ℞ Iodol 3ii, ichthyol, oil of eucalyptus, guaiacol āā ʒi castor oil q. s. to make ʒiv. Ten to twenty drops in capsule three times daily.)

In *chronic peritonitis* absorption and organization should be stimulated by iodin, the use of iodin douches, tampons, Sitz baths, massage, mechanical weight treatment, and Bier's hyperemic treatment. Continued ill-health and loss of earning capacity indicate operative separation of adhesions and pseudo-membranes. Careful plastique, perfect asepsis, avoidance of all unnecessary trauma during operation, and extreme Fowler's position after the operation are essential to success.

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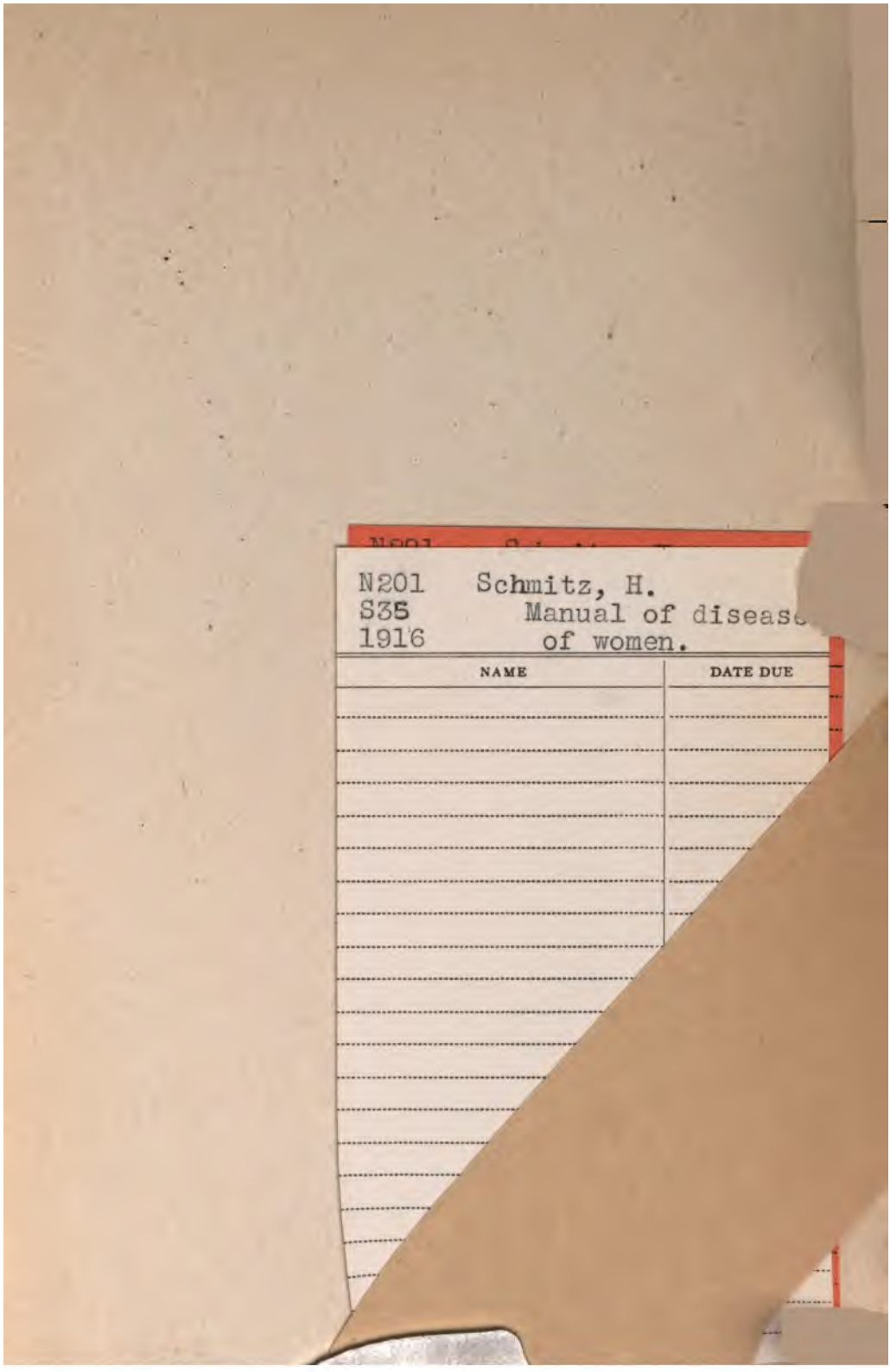
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